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SEVENTY-FIVE YEARS OF PUBLIC HEALTH IN MASSACHUSETTS

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BOSTON

THE seventy-fifth anniversary of the founding of the State Board of Health of Massachusetts calls to mind certain outstanding personalities and their relation to the development of public-health theory and practice in the Commonwealth. Most of these — though not all — were medical men; some of them were distinguished as well in fields other than that of public health. It is the purpose of this article to trace the origin and development of the public-health movement in Massachusetts and to discuss the part played by outstanding men who guided it through the years since 1869.

LEMUEL SHATTUCK, PIONEER

The first to appear on the public-health horizon in this state, of those whose impress was an indelible one, was Lemuel Shattuck. Although bearing a name now famous for its medical connotations, Shattuck was not medically trained, but was first a schoolteacher and later a publisher and bookseller. He early became interested in subjects that are now generally included in the scope of public health. He it was who broke the ground and sowed the seed that many years later produced the first permanent state board of health in America. Shattuck began by obtaining the passage of an act on which is based the present system of birth and death registration in this state. In 1849, he helped to secure the passage of legislation providing for a sanitary survey of the Commonwealth and served as a member of the commission appointed for that purpose. The report of the commission appeared in 1850 and is an extraordinary document, even today, in the light of seventy-five years of rapidly increasing knowledge and experience. The caliber of the thinking exhibited in it may be gathered from the following quotation:

> We believe that the conditions of perfect health, either public or personal, are seldom or never attained, though attainable; that the average length of human life may be very much extended, and its physical power greatly augmented; that in every year, within this commonwealth, thousands of lives are lost which might have been saved; that tens of thousands of cases of sickness occur which might have been prevented; that a vast amount of unnecessarily impaired health and physical debility exists among those not actually confined by sickness; that these preventable evils require an enormous expenditure and loss of money, and impose upon the people unnumbered and immeasurable calamities, pecuniary, social, physical, mental and moral, which might be avoided; that means exist, within our reach, for their mitigation or removal; and that measures for prevention will effect infinitely more than remedies for the cure of disease.

Nearly all the general aspects of present-day health practice are at least foreshadowed in the Shattuck report. As a sampling of the quality and scope of the committee's recommendations one may refer to these: the establishment of a general board of health; increasing the number of effective local boards of health; census of the population; registration of births, marriages and deaths; sanitary surveys; study of sickness in schools; tenements for the laboring classes; quarantine regulations; periodical and special vaccinations; abatement of the smoke nuisance; and many others, some of which have not even yet received adequate attention.

HENRY I. BOWDITCH, ORGANIZER

A period of twenty years followed the presentation of this report, during which the Legislature did nothing to further the objectives of the Shattuck report. Another outstanding figure then appeared in the person of Henry Ingersoll Bowditch. Dr. Bowditch, after graduating from Harvard Medical School in 1832, studied in France under the famous Louis. A characteristic of the latter was his insistence on statistical proof as an aid in estimating the value of the various forms of medical treatment. It is perhaps not too farfetched to suppose that this kind of training influenced Bowditch in his later devotion to the cause of public health in Massachusetts. Bowditch had an independent mind and was not afraid to propose new measures even in the face of disapproval on the part of medical colleagues. He was a man of standing and influence at the time when the State Board of Health was established; he was known for his work on tuberculosis, and had been professor of clinical medicine at Harvard Medical School.

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As a result of energetic effort on the part of Dr. Bowditch and like-minded people and, it is said, because of an outbreak of typhoid fever that aroused fear in the minds of certain legislators, Lemuel Shattuck's project at last took form, and a state board of health was created by act of the Legislature in 1869. Dr. Bowditch was chosen chairman of this board. The statute that started the new board on its long and honorable course has often been quoted but will bear repetition. It reads as follows:

> The Board shall take cognizance of the interests of the life and health of the citizens of this commonwealth. They shall make sanitary investigations and inquiries in respect to the causes of disease, and especially of epidemics and the sources of mortality, and the effects of localities, employments, conditions and circumstances upon the public health; and they shall gather such information in respect to those matters as they may deem proper for diffusion among the people. They shall advise the government in regard to the location and other sanitary conditions of any public institutions, and shall report to the Legislature each year with such suggestions as to legislative acts as they deem necessary.

For ten years, Bowditch remained the mainspring of the State Board of Health, ably seconded by two secretaries of the Board, Dr. George Derby, who died in office in 1874, and his successor, Dr. C. F. Folsom. These ten years were years of real pioneering in the virgin field of public health. The activities of this period have been well summarized by Whipple[1] in these words:

> The Massachusetts State Board of Health was studying Massachusetts, her people and their health, the diseases common among them, the sanitation of their homes, the nuisances to which they were subjected, the water they drank, the method of waste disposal, the ventilation of their school houses, the disposal of their dead. The Board was "taking cognizance of the interests of health and life."

Strange Interlude

In 1879, politics reared its ugly head. General Ben Butler was fulminating against what he termed the numerous commissions that were "spending wastefully the people's money." Those in charge of the government at that time thought to avoid trouble by a little appeasement. The State Board of Health was merged and at least partly submerged in the State Board of Health, Lunacy and Charity. Dr. Bowditch soon resigned in protest.

In the midst of the tribulations of the triple-headed board, a new figure appeared as a worthy successor to Dr. Bowditch. This was Dr. Henry Pickering Walcott, who was later to reach the stature of "a great sanitary statesman." Dr. Walcott joined the Board of Health, Lunacy and Charity in 1880 as executive for its health committee. Two years later he resigned this position to become chairman of the committee. He remained in charge of the State's health activities for thirty-two years.

Henry P. Walcott, Sanitary Statesman

It is hard to appraise the full value of the work of a man like Henry P. Walcott. Always an austere and dominant personality, he nonetheless was able to work with other strong men. A noteworthy example of this was his long association with the engineer, Hiram F. Mills, who shared with Walcott much of the credit for the Board's accomplishments during twenty-eight years, the services of both men ending with the reorganization of the Board in 1914.

It seems, in retrospect, as though Dr. Walcott's strength lay in an ability to take a broad view of the whole field of public health, to appreciate the value of new discoveries and to keep clearly in mind the proper relations of state to local communities. During his long administration occurred some of the great advances in public-health knowledge. These were at once absorbed by the Massachusetts State Board of Health and put to work. Many of these contributions to knowledge originated with the state organization itself. An example of this at once comes to mind in the research work of the Lawrence Experiment Station and the construction of the Metropolitan Water and Sewerage systems.

The Lawrence Experiment Station was planned by Mills in 1887 and represented a great advance in the possibilities of the sanitary control of the environment. It has been said, "The investigations at the Lawrence Experiment Station laid the foundation for the scientific treatment of sewage and have given the initiative for similar investigations in this and other countries."[2] In 1893, a sand-filtration plant for the purification of the water supply of the city of Lawrence was built after plans by Mills. The saving of lives brought about as a result of these and similar engineering accomplishments can hardly be overestimated. The period covered by the life of the State Board of Health from 1869 to 1914 may truly be called the era of sanitary engineering, and of this era two of the outstanding promoters were Walcott and Mills.

There were other well-known figures as well whose work centered in the State Board of Health during that period. Only passing reference can be made to them here. The first in point of time was Dr. Samuel Abbott, secretary of the Board from 1886 to his death in 1904. His report "Vital Statistics in Massachusetts from 1856 to 1895" was a striking piece of work. It may be found in the 1896 report of the Board. The second, Dr. Theobald Smith, had, of course, an international reputation quite apart from his services with the Board. He came to Massachusetts in 1895 as director of the recently established Antitoxin Laboratory. The production and distribution of diphtheria antitoxin represented a new service to Massachusetts physicians and their patients — a type of service that has been steadily extended since then. The Bacteriological Laboratory, too, starting at that time modestly under the State Board of Health, has, through the devoted service of its present chief and her associates, become indispensable to thousands of physicians throughout the Commonwealth. The

Wassermann Laboratory, although established later, in 1915, should be referred to here. It also has grown amazingly in activities and usefulness.

Dr. Smith resigned the directorship of the Antitoxin Laboratory in 1914 and was succeeded by Dr. Milton J. Rosenau, whose great reputation, like that of Dr. Smith, came from research, teaching and writing. Dr. Rosenau held the directorship until 1920.

Before turning from the State Board of Health to its successor, two other secretaries of the Board must be mentioned. Dr. Charles Harrington served from 1905 to 1909, at the same time serving as professor of hygiene at Harvard Medical School. He was succeeded by Dr. Mark Richardson, who ably carried on the work until the Board was reorganized in 1914.

The Department of Health

The year 1914 marks a great change in state health administration in Massachusetts. The State Board of Health had done a magnificent piece of work with the older type of setup, but a different kind of administrative plan was beginning to be popular. Instead of unpaid boards with paid secretaries, states were turning to a single-headed department, with a full-time commissioner and a part-time advisory council, the latter having no executive authority. This kind of health department was proposed to the Massachusetts Legislature and after much discussion the bill was passed. America's first fully functioning state board of health came to an end and was succeeded by the State Department of Health. The man who did the most to get through the Legislature the bill providing for this department was Dr. Enos H. Bigelow, afterward president of the Massachusetts Medical Society, who was at the time a member of the Legislature.

Allan J. McLaughlin and His Staff

It goes without saying that the prime necessity in getting a new enterprise started is the right kind of person to head the organization. Massachusetts was most fortunate in getting just such a one in Dr. Allan J. McLaughlin. A man of forceful personality and extremely wide experience in public health, he had the executive ability necessary for rebuilding an old organization without in any way impairing its essential strength. Dr. McLaughlin came to Massachusetts on leave from the United States Public Health Service. Unfortunately, the exigencies of World War I necessitated his recall to Washington before the term for which he was appointed was up. Nevertheless, before he left he had placed the Department of Health firmly on its feet and, to a large extent, had charted the course it has followed ever since.

The organization of the Department of Health, as has already been indicated, consisted of a full-time commissioner, "skilled in sanitary science and experienced in public health administration," and a public-health council consisting of the commissioner *ex officio* and six members appointed by the governor. The executive and administrative functions were centered in the commissioner, whereas the council had for its functions the making and promulgation of rules and regulations, the taking of evidence in appeals, the holding of hearings and the consideration of plans and appointments required by law. Certain divisions were established, and a system of full-time district health officers was set up.

One of the faults of the old State Board of Health had been its excessive centralization. This was remedied in the new department by the establishment of six divisions, each with a director, who, with one temporary exception, was a full-time worker. These divisions included sanitary engineering, water and sewage laboratories, food and drugs, communicable diseases, biological laboratories and hygiene. In addition, eight health districts were created, with a full-time medical health officer in each. These men were placed in the Division of Communicable Diseases for administrative purposes, but were expected to represent the commissioner in their own districts. This feature of the Department was not entirely new, since a somewhat similar system had been in effect under the State Board of Health. Beginning in 1898 with one man, there were later fifteen inspectors of health, who, however, were required to give only part of their time to this work. Much of the time of these early inspectors of health was devoted to factory inspections. With the creation of the State Board of Labor and Industries, this responsibility was transferred to the new board.

Important as were the duties of the state district health officers, there was a lack of adequate assistance. As a rule there was no local office provided and no clerical assistance. Clerical work had to be done during intervals of visiting boards of health, investigating epidemics, getting materials for case records, looking up all cases of ophthalmia neonatorum and carrying on educational work in the various communities of the district.

Communicable Disease Then and Now

As an example of the tremendous problem that faced the Department in investigating communicable diseases, one may refer to an early statement of the new commissioner, allocating the annual deaths from preventable diseases in Massachusetts to the various divisions best prepared to aid in their prevention. The Division of Communicable Diseases, for example, in 1915 was charged with 15,920 deaths, which included those from syphilis, tuberculosis, pneumonia, diphtheria, measles, scarlet fever, typhoid fever, whooping cough and influenza. Later, owing to other arrangements for handling these diseases, syphilis and tuberculosis were removed from this list, leaving a total of 7920 deaths

within the province of the Division of Communicable Diseases. It goes without saying that a detailed investigation of each of these deaths would have been utterly impossible; only epidemics as a rule could be carefully inquired into. It is interesting to note that if the deaths from these same diseases had been reallocated at the beginning of 1944, the total number would have been 1020 instead of 7920. This great change in the number of annual deaths cannot, of course, be charged to the credit of the Department alone. It does, however, represent the results of the combined efforts of local and state departments of health in co-operation with the medical profession and a far better-informed public.

When the Department of Health was created, all its efforts toward reducing the incidence and death rate of tuberculosis and of the venereal diseases were centered in the Division of Communicable Diseases. Later, however, a Division of Tuberculosis was created, and much later still a Division of Venereal Diseases. In the case of tuberculosis, shortly after 1915 it was felt that better provision should be made for the hospitalization of open cases and the detection of early cases through local dispensary service. This sentiment resulted in legislation establishing county tuberculosis hospitals and extending local dispensary service. Regarding the problem of venereal diseases, which up to the time of World War I had never been adequately discussed, to say nothing of being adequately treated, a great deal of study was given to ways of remedying this condition. A plan was worked out for the reporting of venereal disease by physicians directly to the Department by means of numbers, identification by name being left as a last resort in careless or recalcitrant cases.

Another difficult problem attacked in these early years was that of milk sanitation. Milk at that time was a subject of heated controversy. Epidemics of septic sore throat were far from infrequent, yet the simple expedient of pasteurization of milk on the scale on which it is now practiced was merely a hope for the future. A valuable study of the whole question was made by a milk board made up of members of the Department, and served its purpose in bringing about a better understanding of the whole question.

Personal and Community Hygiene

The period between the establishment of the State Board of Health in 1869 and its reorganization in 1914 may rightly be called primarily the era of sanitary control of the environment. This was the period of the great progress in water and sewage purification already referred to. At the same time strides were made in the direction of adequate supervision of foods and drugs.

Beginning, however, in 1915, a determined effort was made under the leadership of Dr. McLaughlin to carry to the average citizen as much as possible of the knowledge and practices already familiar to public-health workers, in order that the measures initiated by health officials might be better understood and receive more adequate support. Certain aspects of this educational work had to be taken to the medical profession as well, particularly in the case of ophthalmia neonatorum. At the beginning of this period, cases of ophthalmia neonatorum were extremely frequent and blindness often resulted. Years of investigation of each case of sore eyes in newborn babies and the instigation of proper treatment resulted in the almost complete disappearance of blindness due to ophthalmia neonatorum.

Infant mortality at that time judged by present-day standards was startlingly high. Approximately 10,000 infants under one year of age were lost every year in Massachusetts, as compared with about 2700 in recent years. The Department undertook to instruct parents in the simple procedures of infant hygiene and to get prospective mothers to their family physicians for prenatal care.

The need for this kind of instruction led to the first use of public-health nurses. Early in the life of the Department the Division of Hygiene employed two nurses as health instructors. Gradually the number of public-health nurses increased, and the scope of their work broadened until it became one of the most important phases of the Department's work. A beginning was also made in more highly specialized health instruction through the appointment of a health instructor in foods.

One of the most outstanding of all the special efforts directed against infant and maternal mortality was inaugurated when Dr. McLaughlin was able to bring about an alliance between the Department and certain outstanding private agencies whereby it was found possible to get together a group of eight public-health nurses whose salaries were paid by money received from the American Red Cross. Expenses were carried by the Department. A Child Conservation Committee was appointed by the commissioner, chosen from members of the Department and assisted by a large and distinguished advisory committee made up of outside specialists. Under the direction of this committee careful and detailed studies were made in each town in the Commonwealth, and after careful consideration by the Child Conservation Committee these were translated into definite recommendations to the proper authorities of these towns.

Eugene R. Kelley, the Second Commissioner

In 1918, to the great regret of the Department and all interested in it, Dr. McLaughlin was recalled to Washington for war duty and was succeeded as commissioner by Dr. Eugene R. Kelley, who had served with Dr. McLaughlin from the beginning as director of the Division of Communicable Diseases.

Dr. Kelley was at the time a relatively young and vigorous man who before coming to Massachusetts had served as health commissioner of the State of Washington. Imbued with the principles for which Dr. McLaughlin stood, he took up the work where the latter had left it, and carried it forward with the changes that increasing public-health knowledge and increasing public-health consciousness made desirable and possible.

The first great change came in 1919 when the Legislature decided to do away with the large number of boards and commissions that had hitherto functioned in a somewhat overlapping manner and to set up in their place a far smaller number of more responsible departments. In this reorganization the Department of Health fared extremely well. Its name was changed to that of the Department of Public Health, and it received as part of the new Division of Tuberculosis the state sanatoriums hitherto under the jurisdiction of a board called the Trustees of Hospitals for Consumptives. This division was later to have another institution placed under its care, namely, the new Cancer Hospital at Pondville.

In 1929, an outstanding attempt was made under the inspiration of Dr. Henry D. Chadwick, afterward commissioner of public health, to reach tuberculosis at its source through the early detection of the disease in children. With Dr. Kelley's backing a ten-year tuberculosis program was begun; it was usually referred to as the Chadwick Clinics and was named, of course, after the man who conceived the idea and carried it to completion.

One of the problems of a good administration has always been to get those who are interested in a given field to co-operate adequately. The great field of the health of the child has always been of interest to the people of the Commonwealth. School medical inspection began as early as 1894 in Boston, and later legislation required medical and nursing supervision of all Massachusetts school children. Two state departments in the very nature of things are deeply concerned with the health of the school child, namely, the Department of Public Health and the Department of Education. Yet for many years there was little effective co-operation between these two departments. Dr. McLaughlin was deeply concerned over this, and shortly after Dr. Kelley succeeded to the commissionership a close relation was established, whereby both departments united to stimulate local interest in the school health problem and to aid local authorities in strengthening and increasing the work already in operation. The possibilities of such co-operation are unlimited.

As time went on the need was felt for additional specialization. Increased knowledge of nutrition and of the need of doing something to cut down the incidence of the universal disease, dental caries, led to the appointment by the Department of workers who specialized in these fields and who could serve as consultants to the more generalized staff workers as well as to local organizations. Consultants in public health nursing stimulated to a remarkable degree the establishment of new public-health nursing services in the local communities.

Early in the history of the Department of Health, it was felt that something should be done toward reducing the cancer death rate. Not much seemed possible at that time, but it was believed that it would be a step in the right direction if a free tumor diagnostic clinic were established. This was done in 1919 through the co-operation of the Harvard Cancer Commission. In 1925, the Legislature directed the Department of Public Health and the Department of Public Welfare to make a study of the cancer situation in Massachusetts and to recommend necessary measures for successfully attacking this problem. Dr. Kelley's death occurred before this study was completed, but under his successor a report was made to the Legislature, and as a result money was appropriated for a hospital to be devoted to the treatment of cancer patients. This cancer program has grown until it now represents the larger part of the activities of a special division in the Department, which has carried on extensive statistical studies and a great deal of public education, seemingly with demonstrated success.

George H. Bigelow, Public-Health Evangelist

On the death of Dr. Kelley after eight years' service as head of the Department, his deputy, Dr. George H. Bigelow, was chosen commissioner. With a brilliant mind and an energetic temperament, Dr. Bigelow threw himself wholeheartedly into the problems confronting the Department, struggling against public inertia, sometimes with indignation and at other times with a kind of sardonic humor that included both the obstructionist and himself. The measures proposed by Bigelow were as a rule sound, and have been or will ultimately be accepted as good public-health practice. If all milk were pasteurized, unquestionably Massachusetts would be a safer place in which to live. Even now, as a result of the insistence of Bigelow and his successors and other like-minded persons in and out of the Department, about 80 per cent of the population of this state live in communities that require that the milk supply be pasteurized or certified. Over 90 per cent of the milk consumed in the Commonwealth is pasteurized. Many of the other advances made during Dr. Bigelow's commissionership are referred to elsewhere.

After eight years of active leadership, Dr. Bigelow left the Department to become director of the Massachusetts General Hospital and was succeeded by Dr. Henry D. Chadwick, who had been associated with the Department of Public Health for many years as superintendent of the Westfield Sanatorium, director of the Division of Tuberculosis

and guiding spirit in the promotion of the childhood tuberculosis program.

Later Milestones

At this point we shall have to take leave of personalities, since all those who have been connected with the later activities of the Department are still alive. The development of the Department of Public Health has continued and parallels the general advances in public health throughout the country and the world.

One of the later developments of considerable interest was the study made in 1935 and 1936 by the Massachusetts State Health Commission. This commission was appointed by the governor and was made up both of members of the Department and of outstanding medical and public-health experts from outside the Department staff. The commission studied existing health laws and practices and made suggestions for legislation that would anticipate normal expansion for years to come. The Legislature accepted some of these suggestions, which have been translated into statutes.

The year 1934 saw the ending of the ten-year tuberculosis program. From that time on the work has been carried on in a more or less decentralized way through county and state sanatoriums. Actual or suspected cases of tuberculosis in children are followed up through examinations by a state clinic group. During the period of the demonstration program about 400,000 school children were examined, and the importance of the prevention of tuberculosis has been brought home to practically every family in the Commonwealth.

In 1935, a pneumonia study that had been begun in 1931 with the financial support of the Commonwealth Fund of New York City was concluded. At that time the Department assumed the expense of preparation and distribution of serum for the treatment of this disease. Three books on pneumonia were written as part of this study, one a handbook for physicians on the treatment of pneumonia.

Experimental work was carried on with reference to the preparation and evaluation of placental extract in the control of measles. This was financed by the Commonwealth Fund, and the work was done by the Harvard Medical School through the Children's Hospital and in co-operation with the Department of Public Health.

During this year also the cancer work was extended by the legislative provision for a cancer and tuberculosis unit to be built on the grounds of the Westfield State Sanatorium.

The year 1936 was noteworthy for the beginning of participation by the Department of Public Health in the activities provided for in the federal Social Security Act. It is difficult to estimate the potentialities for the future of this type of federal and state co-operation. It brought and continues to bring to the Department large sums of money, both through federal grants and through state appropriations, and involves a great expansion of both personnel and activity. The latest example of this is the Emergency Maternal and Infant Care Program for the wives of certain men in the armed services of the United States. Other specific examples of the use of Social Security funds are the programs for the care of crippled children and the control of venereal disease. It is altogether too early to assess accurately the future possibilities or, for that matter, the drawbacks in this type of joint action.

In 1937, under the provisions of an act of the Legislature, the Department of Public Health obtained funds for the hospitalization of a limited number of cases of chronic rheumatism. This work has been carried on under a contract with the Massachusetts General Hospital.

In 1939, because of the appearance of equine encephalomyelitis, the Department carried out an extensive study of the mosquito situation in Massachusetts as an aid in reaching some determination as to the way in which this disease is spread. Definite conclusions will have to await further opportunities for study.

One of the newer efforts to raise the standards of public-health practices stemmed from the recommendations of the Massachusetts State Health Commission, already referred to, and had to do with the approval of bacteriologic and serologic laboratories by the Department of Public Health. Under the provisions of this statute such laboratories may apply to the Department for approval. This is purely voluntary on the part of laboratories, except in the case of those that wish to perform serologic tests for syphilis in cases of pregnancy and those wishing to qualify for performing premarital examinations. This work is going on quite satisfactorily and is meeting with a good response from the laboratories.

In 1941, legislation was passed under which hospitals are licensed by the Department of Public Health in accordance with regulations promulgated by the Department. Provision for licensing dispensaries had been made many years previously.

The Department of Public Health has now reached the point where, because of its size and its varied activities, considerably more decentralization is necessary. In view of the traditional policy of the Commonwealth to delegate in so far as possible authority in public-health matters to the local boards of health of the various municipalities, large and small, it is more or less imperative that these communities, especially the smaller ones, be able to obtain easily advice and assistance from the Department. From the earliest days of the State Board of Health, assistance of this sort has been offered, and as time has gone on, more and more facilities have been provided for this purpose. The establishment of the district-health-officer plan marked a great increase in efficiency on the part of the Department of Health. It has been found, however, that a

further step must be taken involving the strengthening of the district offices and the placing under them of all types of services that the Department has to offer to the local communities. This strengthening is progressing at a steady rate. By this and other means, it is confidently expected that the Department will be in a position to meet the many changes that the future will undoubtedly bring in the field of public-health administration.

Summary

Seventy-five years ago, in 1869, Massachusetts established a state board of health. This was the first unit functioning from the beginning as a state board of health. It came about after twenty years of discussion following the presentation to the Legislature of a remarkable document on sanitation, largely written by Lemuel Shattuck.

The Board of Health functioned as such from 1869 to 1879 and then was merged in the State Board of Health, Lunacy and Charity. This combination lasted for seven years, but in 1886 it was seen to be desirable that the Board of Health be made independent once more.

From 1886 to 1914, the Board of Health became increasingly necessary as public-health knowledge increased; it not only played an honorable part but added greatly to the knowledge of its time. In 1914, the board was transformed into the Department of Health, with responsibility centered in a commissioner of health. In 1919, the name was changed to the Department of Public Health, and as such it has continued to the present time.

An attempt has been made to show the progress of the public-health movement in Massachusetts by discussing the accomplishments of certain outstanding men, beginning with Henry Ingersoll Bowditch and following down through the years with Dr. Henry Pickering Walcott, Mr. Hiram F. Mills, Dr. Samuel Abbott, Dr. Allan J. McLaughlin, Dr. Eugene R. Kelley, Dr. George H. Bigelow and their successors.

A constant advance is evident, conservative but steady and paralleling the best being made in the rest of America and throughout the world. The Department is now at the height of its efficiency and opportunity for service to the people of the Commonwealth, and is looking forward to taking its part in the still greater public health that undoubtedly lies ahead.

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ACUTE PHOSPHORUS POISONING*

Report of a Case with Recovery

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SOMERVILLE, MASSACHUSETTS

AT ONE time phosphorus was a common poison owing to the use of yellow phosphorus in match tips. This practice is now prohibited by law, and as a result the number of deaths from phosphorus poisoning has been greatly reduced.[1] Phosphorus is, however, still used in various forms of rat and roach pastes that are easily available to the pica of some children in certain environments. Several deaths of children have been reported from eating fireworks, especially the so-called "spit devil" variety.[2] The majority of cases of phosphorus poisoning in adults are the result of suicidal attempts, some form of rat paste or vermin killer containing 1 to 4 per cent phosphorus being used. Cases have been reported in which phosphorus was used as an abortifacient.[3]

Toxic Dose

The toxicity depends to a great extent on the physical state of the phosphorus, which governs the rate of absorption, as well as on personal peculiarities, those who are the subject of nervous diseases probably being somewhat more susceptible than others.[2] Death has occurred after the ingestion of match heads estimated to contain 65 to 130 mg., but recovery has occurred after the taking of about 400 mg. In one of Smith's[1] cases, match heads made of yellow phosphorus were enclosed in sweets and used for homicidal purposes; although a sweet containing 200 mg. of phosphorus was taken, the patient recovered after severe symptoms.[1] The consensus is that a dose of 50 to 65 mg. will produce decidedly dangerous or fatal symptoms in a healthy adult.[2]

Symptoms and Signs

There are two phases in the symptomatology — a primary one due to local irritation of the gastrointestinal tract, and a secondary one due to the action of the absorbed poisons.

The primary symptoms are usually delayed for two to six hours following ingestion. They consist of abdominal pain, burning, nausea, vomiting and diarrhea. The breath and vomitus have a characteristic garlic odor, and the vomitus is frequently

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luminous. Thirst is usually intense, and gaseous eructations are frequent. Diarrhea is a variable feature, occurring in about one third of the cases; when it is present the feces may be dark and luminous.

If the patient does not succumb to shock from the immediate effects of the phosphorus, there ensues a period of two or three days when the patient shows marked improvement, following which the second stage of poisoning is heralded by nausea and by protracted emesis, which produces a blood-streaked vomitus, diarrhea and abdominal tenderness in the region of the liver. The conjunctivas and the skin become jaundiced. There are headache, weakness and malaise. There may be bleeding from the nose or vagina, and hemorrhagic spots may appear under the skin and mucous membranes. The urine becomes scant and highly colored and contains blood, droplets of free fat and albumin; the urea content is lessened, and amino acids, especially leucin and tyrosin, are present.[2] Lawrence and Huffman[4] call attention to an increase in the number of monocytes in the blood. Marked leukopenia has never been reported, so far as is known, but in the case here reported the white-cell count was 1200 on the day of admission. There is usually a definite decrease in the blood sugar, which is explained by failure of glycogenolysis, owing to liver destruction, to check the downward course of the sugar level.[5] The liver is usually enlarged. There is frequently slight fever, the pulse becomes rapid, weak and irregular, and the patient gradually sinks, dying within about a week after ingestion of the poison.[1]

Pathology

It is clear that the post-mortem changes vary with the duration of the poisoning. The skin is jaundiced, and hemorrhagic spots may be visible over the skin. On opening the body a garlic odor may be noted, and in acute cases the stomach contents have a faintly luminous appearance. The mucous membrane of the gastrointestinal tract shows inflammatory reaction and occasionally small erosions. Patches of submucous hemorrhage may be observed. Phosphorus is one of the most potent causes of fatty degeneration, and this change is present in marked degree in the liver, kidneys, heart and even the voluntary muscles.[6] The nervous system is also vulnerable to phosphorus. The inferior olivary bodies and the lipophilic cells are involved early and account for the symptoms referable to the central nervous system.[7]

The liver shows the most characteristic changes; it is usually enlarged and of a lemon-yellow tint, it feels greasy and soft, and small hemorrhages may be seen on the surface and in the substance. Microscopically, extensive fatty degeneration of the liver cells is observed, the accumulation of fat in Kupffer's cells being an early manifestation.[4] The kidneys are larger than normal, icteric in color and of a very soft consistence. They also show marked fatty degeneration microscopically.

Phosphorus may be detected in its unoxidized form for several days, and it has been found in the body after burial for a month. Scherer's test is a good preliminary one, but since it is not specific and is affected by a variety of reducing agents, it is of value only in proving the absence of phosphorus or in indicating the desirability of other, more specific tests. The most reliable test is the Mitscherlich method, which depends on separation of free phosphorus in a current of steam, where it phosphoresces. Both these tests are described in Smith's *Forensic Medicine*.[1]

Case Report

D. C. (K-1889), a 23-year-old, Italian schoolteacher, entered the Newton Hospital on March 3, 1944, and was discharged on March 28. The diagnosis was acute phosphorus poisoning. The past history and family history were noncontributory. Four days prior to admission, following an argument with her fiancé, the patient ingested more than a teaspoonful of a rat and roach paste that contains 2.5 per cent phosphorus and 97.5 per cent inert matter. The amount of phosphorus ingested was estimated at about 130 mg. Because of fear the patient consulted a local physician an hour later, and he suggested that milk be taken immediately. Two glasses of milk were then ingested, followed half an hour later by a drink of whisky. Three hours after ingestion of the poison the patient began to have a headache, felt dizzy and nauseated and had epigastric pain. There were frequent attacks of vomiting during the night, the vomitus appearing "bright yellow and glowing." The vomiting relieved the abdominal pain for a short time. The mouth became extremely dry. The abdominal pain soon recurred and was severe enough to cause groaning. A severe headache developed and persisted up to admission. The following night the patient felt improved enough to attend a dance. Two days later she became so dizzy and weak that she was forced to remain in bed. Marked nausea returned, with occasional vomiting, and the abdominal pain also returned. The urine became highly colored and somewhat reduced in amount. A physician advised immediate hospitalization.

Physical examination revealed an alert young woman lying in bed in no apparent acute distress. There was a faint lemon-yellow color to the skin. The conjunctivas were icteric. The pupils were equal and reacted to light. There was a small, dried, bloody crust in the right nostril. The mucous membranes of the mouth were somewhat pale. No petechiae were present. The tongue was dry, with a gray coating. The neck was not rigid, and a few nontender cervical lymph nodes were palpated bilaterally. The lungs were resonant and clear. The heart sounds were good, with a regular rhythm and a rate of 90. A soft systolic murmur was heard over the precordium and was maximum at the apex. There was moderate tenderness to palpation in the right upper quadrant of the abdomen. The liver could not be palpated. No masses were felt, and the remainder of the examination was essentially negative. The temperature was 99°F., the pulse 80, and the respirations 15. The blood pressure was 110/70. The urine showed a specific gravity of 1.022, no albumin and complete reduction of Benedict's solution (the patient had just received 1000 cc. of 10 per cent glucose solution parenterally). The sediment contained 2 to 4 red cells per high-power field and no leucine or tyrocine. The red-cell count was 4,200,000 and the hemoglobin 86 per cent. The white-cell count was 1200, with 24 per cent segmented neutrophils, 8 per cent young neutrophils and 68 per cent lymphocytes.

The day following admission, the jaundice was more evident and the patient complained of a garliclike taste in the mouth. She was still nauseated but was able to take fluids. There were two episodes of nosebleed from the left nostril. The nonprotein nitrogen was 47.7 mg. per 100 cc., the icteric index 37, and the bilirubin 9 mg. (direct). The prothrombin

time was 28 seconds (normal, 18 seconds). The urine revealed a slight trace of albumin, with 60 to 100 red cells per high-power field. A bile test was positive, and urobilinogen was positive in a 1:30 dilution. For the following 2 days the patient felt generally improved, with no complaints except tightness in the chest, tingling in the veins and slight dizziness following Pentnucleotide therapy for the leukopenia. The jaundice, however, had definitely increased. The liver was felt 2 fingerbreadths below the right costal margin.

Five days after admission, the icteric index had risen to 111, and the white-cell count to 5000. The urine was still negative for tyrocine and leucine crystals. An electrocardiogram revealed a sinoauricular tachycardia, with an occasional extrasystole. There was no evidence of myocardial damage. The following day the jaundice began to clear, and from then on the patient made an uneventful recovery. On the 18th day, the jaundice had cleared except for a slight icteric tinge to the conjunctivas. The prothrombin time was 20 seconds. A bromsulfalein test was normal. The serum bilirubin was 2 mg. per 100 cc., and the icteric index 18. The white-cell count was 9400 and the red-cell count 4,200,000 with a hemoglobin of 84 per cent. The patient was discharged improved on March 24 with instructions to return to the Outpatient Department for a follow-up.

Treatment consisted of a high-carbohydrate, low-fat diet, with daily intravenous infusions of 10 per cent glucose in saline solution. Twenty units of regular insulin was administered intramuscularly prior to the intravenous infusions. One gram of calcium gluconate was administered orally three times daily. One hundred milligrams of thiamine hydrochloride and 200 mg. of ascorbic acid were administered intramuscularly daily. For the marked leukopenia 10 cc. of Pentnucleotide was given intramuscularly three times a day for 3 days, twice a day for 4 days, once daily for 4 days, and then once every other day until the white-cell count reached 9000. The urine was maintained at pH 7.5 with sodium bicarbonate by mouth.

In addition to the treatment in this case, when patients are seen early every attempt should be made to prevent the absorption of orally ingested phosphorus. Gastric lavage should be performed even if the patient is first seen several hours after the poison has been taken. A suitable chemical antidote is copper sulfate (0.25 gm. in a glass of water), the effect of which is to envelop the phosphorus particles with an insoluble coating of copper phosphide. Oxidizing agents, such as potassium permanganate (1:1000 solution) and hydrogen peroxide (2 per cent of the official preparation), may aid the conversion of elemental phosphorus to harmless oxidation products. Following gastric lavage, a cathartic of magnesium sulfate should be administered to cleanse the bowels.[8]

* * *

Phosphorus is a severe destructive poison. It is rapidly absorbed through the gastrointestinal tract and manifests itself by fatty degeneration and infiltration of the brain, liver, kidneys and practically all the other viscera of the body. The early symptoms are usually those of insult to the central nervous system and those due to local irritant action, as in the gastrointestinal tract. Later the symptoms are those of liver destruction, with jaundice, enlargement of the liver, an increased icteric index and usually hypoglycemia. Most cases of phosphorus poisoning have terminated fatally; hence, it is essential to make a diagnosis as soon as possible and institute treatment immediately. These cases should always be considered as emergencies.[9]

Summary

Acute phosphorus poisoning is described from the standpoint of toxicity, symptomatology and pathology.

A case of acute phosphorus poisoning due to ingestion of a rat paste, with an uneventful recovery, is reported.

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MEDICAL PROGRESS

HEMATOLOGY

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ALTHOUGH anemia is by no means all of hematology, it is often considered its most important phase. This year's review will be limited to a more or less personalized discussion of this subject.

GENERAL CONSIDERATIONS

Classification

The terms "secondary anemia" and "primary anemia" must by now be considered as clearly outmoded. Since anemia is always a symptom of some underlying disorder, all anemias are necessarily secondary. If this is the case, there is no point in discriminating between primary and secondary cases. The classification of anemia on the basis of color index, cell volume or cell diameter, or all three, has become deservedly popular, chiefly because it gives orientation regarding the possible etiologic and therapeutic factors in a given case. Thus hypochromic microcytic anemia is usually indicative of chronic iron deficiency and of a probable therapeutic response to iron rather than to liver. These features are listed in Table 1.

For a complete classification, cell size and color index are, however, not enough; the important consideration is the *cause* of the anemia. In every case, it is essential, preferably before therapy is begun, to attempt a definition of etiologic factors. This can generally be done if it is remembered that anemia is usually due to one of three factors: a deficiency of materials requisite for blood formation; a disturbance in the marrow; and an increased loss of the bone-marrow's finished product, the red cell, either through hemorrhage or increased hemolysis.

The deficiency anemias are caused by a variety of factors: a dietary that is deficient in iron, protein or vitamin B complex; an inadequacy of the gastric juice, which is deficient in either free hydrochloric acid or Castle's enzyme ("intrinsic factor"); chronic intestinal malabsorption, as in chronic diarrhea, sprue and so forth; hepatic disease, in which the factor of storage is affected; and multiple pregnancies, in which the developing fetus absorbs an exceptional amount of material from the mother. Single etiologic factors are by no means as important in this regard, however, as multiple causes. Thus a woman on an inadequate intake of iron does not usually develop anemia unless she also has a gastric disturbance (achlorhydria) and either many pregnancies or menorrhagia. And an individual with sprue probably not only has a deficient diet but a chronic intestinal disturbance as well.

Anemia due to a bone-marrow disturbance is normochromic and normocytic because, although red-cell production is more or less seriously impaired, the red cells that are produced are of normal size and hemoglobin content. There is likewise an associated leukopenia and thrombocytopenia, — that is, pancytopenia, — since if the bone-marrow is the site of involvement, *all* its elements are usually affected. Numerous disturbances of the bone-marrow itself are known: aplasia and hypoplasia, as with benzol and other types of chemical poisoning and the hypoplasia of myxedema and of chronic nephritis with azotemia; replacement by leukemia, lymphosarcoma, multiple myeloma and other tumors; and diminished or inhibited delivery, as in hypersplenism (see below). The bone-marrow biopsy, whether by puncture or trephine, is of the utmost value in the differential diagnosis of these conditions.

TABLE 1. *Classification of Anemias According to Cell Size, Color Index and So Forth.*

TYPE OF ANEMIA	COLOR INDEX	CELL DIAMETER (M. C. D.) *microns*	CELL VOLUME (M. C. V.) *cu. microns*	PROBABLE CAUSE	PROBABLE EFFECTIVE THERAPY
Normocytic	0.9–1.1	7.3–7.8	85–100	Bone-marrow disturbance	Transfusions (?)
Microcytic	0.4–0.8	5.5–6.5	65–85	Iron deficiency	Iron
Macrocytic	Above 1.1	7.8–8.3	100–130	Liver-extract deficiency	Liver extract

Anemia due to excessive blood loss may be either hemorrhagic or hemolytic in type. Severe hypochromic anemia due to chronic slight bleeding from the gastrointestinal tract often remains undiagnosed, and is frequently mistreated, usually by means of continued injections of liver extract. The blood picture of chronic hypochromic microcytic anemia in association with increased polychromatophilia of the red cells, a slight increase in the reticulocytes, polychromatophilic target cells, increased platelets and a slight "shift to the left" of the polymorphonuclear cells is usually due to chronic bleeding and

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not to simple iron deficiency. Anemia due to excessive hemolysis is usually associated with a normal or increased color index, and a normal or increased cell volume in the presence of small red cells, that is, the cells are thicker than normal. Bilirubinemia is present, and the reticulocytes are greatly increased.

From the foregoing, it may be seen that, in any given case of anemia, it is necessary to determine among other things, the factors of color index, cell volume and cell size and the various etiologic factors concerned. The classification that I prefer at present is based on both these criteria. Thus a given case may be classified as follows: macrocytic anemia, inadequate intake of protein, gastric achlorhydria — that is, liver-extract deficiency or pernicious anemia; microcytic, hypochromic anemia, inadequate intake of iron, gastric achlorhydria, profuse menses — that is, chronic hypochromic, iron-deficiency anemia; or normochromic, normocytic anemia, benzol poisoning — that is, aplastic anemia.

Diagnosis

The working out of the etiologic factors in a given case presenting anemia as one of its features may either be simple or quite complex. As in other branches of medicine, one has recourse to every available bit of data derived from the history, the physical examination and the various laboratory tests, including x-ray examination and the sternal bone-marrow biopsy.

From the history one learns about the diet, the matter of gastrointestinal symptoms, the question of bleeding into the skin or from various organs and such miscellaneous symptoms as glossitis, weight loss, pains in bones and fever. The family history is important from the standpoints of anemia, early graying of the hair, jaundice and so forth. When taken with proper regard to possible disease entities, a careful history often suggests the diagnosis.

In the physical examination one pays particular heed to the questions of pallor, icterus, the appearance of the tongue, the matter of enlarged peripheral lymph nodes and the size of the spleen and liver. Careful inspection of the skin and buccal mucous membranes should be made for petechiae, ecchymoses and spider telangiectasia. The remainder of the physical examination is by no means neglected because many so-called "blood cases" turn out to have a general disease, such as myxedema, nephritis or subacute bacterial endocarditis.

The laboratory data required for diagnosis may be extensive, but often a few simple tests of the blood give the probable diagnosis. The hemoglobin estimation should be done either with a well-calibrated hemometer or preferably by means of a photoelectric colorimeter. Red-cell, white-cell and differential counts are ordinarily sufficient, although in many cases it is important to know, or at least to estimate, the platelet and reticulocyte levels. Estimations of these factors are made by noting the extent of polychromatophilia and the abundance of the platelets in the stained smear. The hematocrit is valuable from the standpoint of determination of the mean corpuscular volume and thus of the red-cell size. Bilirubin determinations are important from the standpoint of hemolysis. Many other tests are occasionally brought into play. These include determination of the hypotonic and other fragilities, the question of hemolysins and agglutinins, examination of the stools for occult blood and urobilinogen, liver-function studies and gastric analysis.

In the cases with pancytopenia, no test is of greater importance than a biopsy of the sternal marrow. In most cases, a simple needle aspiration is sufficient to make a positive diagnosis (leukemia, multiple myeloma, hemolytic anemia and so forth). Occasionally, however, removal of a button of bone by the trephine method becomes necessary, particularly when needle aspiration is unsuccessful. This is particularly the case when fibrosis, aplasia or metastatic malignancy is present. By this method the topography of the marrow and the relation of cellular elements to bone, fibrous tissue and so forth can be recognized at a glance. Metastatic nodules, clumps of Gaucher cells and diffuse myelofibrosis are readily recognized. In my own work-up of a hematologic problem, a sternal puncture aspiration is performed as a routine measure; it not only gives diagnostic help but also is of assistance regarding the pathologic physiology of various disease conditions.

Deficiency Anemias

Chronic Iron Deficiency

A continued deficiency in iron results in a shortage of hemoglobin for the developing erythrocytes in the bone marrow. As a result, the normoblasts have inadequate cytoplasm and the mature non-nucleated red cells are small and hypochromic. The anemia, which is hypochromic and microcytic, is by no means the only evidence of iron deficiency, which becomes manifest as well by changes in certain epithelial structures. Thus, the skin becomes wrinkled and flabby, the hair dry and gray, the tongue red, atrophied and shiny, and the fingernails flattened. Indeed, changes of other epithelial structures, including atrophy of the esophageal mucosa with web formation and of the stomach and intestines, often occur.

A state of chronic iron deficiency is brought about in various ways — through a diet inadequate in iron-containing food, by way of gastric achlorhydria, chronic diarrhea, chronic hemorrhage or multiple pregnancies or by a combination of two or more of these factors. Fewer cases of this condition seem to turn up nowadays than formerly, perhaps because incomes are higher and dietary inadequacy is therefore lessened. One should never mistake a chronic "idiopathic" iron-deficiency state for a

reduction in hemoglobin due to chronic bleeding from the gastrointestinal tract; for this reason, studies of the stools for occult blood and careful x-ray examination of the gastrointestinal tract are important.

Chronic hypochromic anemia is far more frequent in women than in men, probably because of the drain of the monthly loss of appreciable quantities of blood with the menstrual cycle, and the loss of hemoglobin-building substances to the fetus in pregnancy. Bethell, Blecha and Van Sant[1] studied the dietary habits and hematologic values of a group of 484 pregnant women in two rural counties of Michigan. Although all the women were from farms and could thus presumably obtain adequate amounts of good food, only a small number met the present-day recommended allowances for pregnancy. The incidence of anemia, almost all of which was of the hypochromic iron-deficient type, was 25.4 per cent. There was a direct relation of the incidence of anemia to the degree of iron inadequacy. As the authors state:

> Although, in adults, a low food intake of iron is not generally an important cause of anemia, an exception must be made in the case of pregnant women with their increased requirements. Moreover, pre-existing stores of iron are essential to meet these added needs. Therefore, it is the quantity of iron consumed before conception that largely determines later evidences of its deficiency.

Hypochromic anemia in young and adolescent women, and for which no cause is apparent, has for centuries been known as "chlorosis." Recently, the presence of complete achlorhydria in a gastric juice of low volume has been recognized as an important feature of the disorder. The lack of hydrochloric acid may result in an insufficient digestion of iron-containing food and thus in hypochromic anemia. Chlorosis has become a rarity, and male chlorosis is almost a curiosity. I have seen only one such case, that of an adolescent boy who had a hemoglobin value of less than 20 per cent and complete achlorhydria; there was rapid improvement with inorganic iron by mouth. Thomson,[2] however, found 9 cases of apparently idiopathic hypochromic anemia in adolescent male recruits. Pallor, a smooth tongue (4 cases), complete achlorhydria (5 cases, with hypochlorhydria in 4), apical systolic murmurs, a barely palpable spleen (4 cases), spooned nails (4 cases) and prompt response to inorganic iron were present.

The nutritional hypochromic anemias of early infancy are rather common, particularly when infants are kept too long on a low iron intake. Kracke and Platt[3] point out that they may be due to such factors as prematurity and disorders of the alimentary tract, including celiac disease, diarrheas and intestinal parasites.

It should be remembered that hypochromia — a low color index — indicates a hemoglobin deficiency in the presence of more or less normally productive islands of red cells. Liver extract, for all its potency in pernicious anemia, has no effect whatever, even of an additive type, in cases of iron deficiency. Its use in such cases has little justification. Curiously enough, many cases of chronic iron deficiency are treated for months with such wholly useless medications as liver extract, vitamin pills and so forth, while treatment with iron is withheld. The place of copper in the treatment of iron deficiency in man remains extremely questionable, although the group at the University of Wisconsin has consistently maintained the value of copper in both rat and human hemoglobin synthesis. In a recent paper, Maass, Michaud, Spector, Elvehjem and Hart[4] present evidence that copper is similarly useful in the hemorrhagic anemia of puppies. The value of copper in the hemoglobin synthesis of milk-fed rats has recently been questioned by Smith and Medlicott,[5] who found that it might stimulate erythropoiesis but did not cause an increase in hemoglobin.

Chronic Liver-Extract Deficiency (Pernicious Anemia)

In this condition, a deficiency in the liver-extract principle is brought about either by a grossly deficient protein content of the diet, by a lack of production of Castle's proteolytic enzyme ("intrinsic factor") by the stomach, by a loss of the liver-extract principle from the body because of malabsorption by the intestines, as in prolonged diarrhea, or by a failure of the liver to store or perhaps to make utilizable the liver-extract principle. Although lack of the proteolytic enzyme is probably by far the most significant factor in the etiology of the disease, it is quite likely in many cases that the disease does not fully develop until some other factor, such as dietary inadequacy or pregnancy, supervenes. The ensuing deficiency in liver-extract principle not only results in a malfunctioning, megaloblastic bone marrow and macrocytic anemia but also in disorders of epithelial structures and the central nervous system. Thus, the hair becomes gray or white early in life, the tongue loses its papillae, and the spinal cord may develop subacute combined degeneration. In fact, many cases with little anemia may show a high degree of neurologic involvement. In pernicious anemia one may discriminate four more or less distinct forms: a purely hematologic type, characterized by severe macrocytic anemia and by little if any neurologic involvement; a fundamentally neurologic type, characterized by combined system disease and by little if any anemia; a refractory type, in which therapy even with huge doses of liver extract is only partially effective (in this type free hydrochloric acid may be present to some extent in the gastric juice — the "achrestic anemia" of Israëls and Wilkinson[6]); and a hemolytic type, in which there is unusually striking jaundice and a large spleen.

That pernicious anemia is a deficiency state cannot seriously be questioned. That it is also, at least to some extent, a hemolytic anemia, is attested by

the acholuric icterus and the high pigment output in the feces and urine. The cause of the hemolytic component remains obscure. Johnson, Freeman and Longini[7] present evidence that the red cells of untreated cases of pernicious anemia are unusually sensitive to the digestive products of fat and speculate that this may be one of the etiologic factors of the anemia. Many authorities believe that the piling up in the bone marrow of large numbers of primitive red cells and their forerunners results in their gradual destruction and thus in the formation of bilirubin. To my mind, this seems a more logical explanation of and one more compatible with the idea that the disease is a pure deficiency state with resultant changes in the bone marrow, epithelial structures and nervous system.

The constitutional nature of the disease is attested by its relatively high incidence in members of the same family (4 to 15 per cent in various statistics), and by its greatest incidence in blue-eyed, fair-haired people and its lowest incidence in brown-eyed, dark-haired people. In Negroes, the disease has been said to be rare. Schwartz and Gore[8] found that this rarity was more apparent than real, at least at the Cook County Hospital in Chicago. There, of 1000 consecutive patients with pernicious anemia entering the hospital from 1931 to 1942, a total of 93 were Negroes. The incidence of the disease in Caucasians admitted to the hospital was 170 per 100,000, contrasting with the rate of 36 in Negroes. It was found that the Negroes tended to show a higher incidence in youth and a lower incidence in old age than did the Whites.

The pernicious anemia of pregnancy is infrequent, but when it occurs, it is liable to be extremely severe and rather markedly refractory to liver-extract therapy. It has a number of atypical features — lack of outspoken jaundice, lack of outspoken macrocytosis, presence of free hydrochloric acid in the gastric juice, no neurologic phenomena and slow response to liver extract.[9] In a recent case, the patient walked into my office with a hemoglobin concentration of 14 per cent (2.2 gm.) and a red-cell count of 800,100. The bilirubin value was only 0.5 mg. per 100 cc., and the red-cell picture was by no means characteristic. The striking thing in the blood picture was the presence of numerous multilobulated polymorphonuclear cells (so-called "pernicious-anemia neutrophils"), and this was by far the best single clue to the diagnosis, which was substantiated by puncture of the sternal marrow. Response to massive therapy with liver extract was slow and was considerably aided by the use of several transfusions. The various clinical and laboratory features of this condition have been fully presented in a recent study by Callender.[10] This author points out that the theory of a temporary intrinsic-factor deficiency during pregnancy does not fit in with all the facts related to the anemia, especially since liver extract is frequently ineffective. She also emphasizes the atypicalness of the blood picture and the great value of the sternal puncture in diagnosis.

Carter and Traut[11] emphasize the cardiovascular manifestations, which they state occurred in 257 of 300 cases of pernicious anemia. This figure, however, is considerably invalidated by the inclusion of "weakness" as a cardiovascular symptom and its occurrence in 231, or 77 per cent, of the cases. The authors' conclusion that all the usual criteria of cardiovascular disease may occur solely as the result of anemia is, however, distinctly valid, together with the statement that these manifestations disappear after treatment with liver extract.

The symptoms of cord involvement in pernicious anemia are well known, but those relating to the brain are often unrecognized. The patient is frequently suspicious and even paranoid. I have seen a number who became psychotic as they grew older. Some of the most profound examples of neurasthenic disability are seen in this group of cases. Adams and Kubik[12] studied the histology of the central nervous system in 2 cases of pernicious anemia and found that the cerebral and spinal-cord lesions were almost identical, consisting of a more or less diffuse though uneven degeneration of the white matter. Attempts have been made to explain the neural symptoms of the disease as due to a deficiency differing from that which causes the anemia. At least in human beings, no such differentiation has been demonstrated. In swine, Wintrobe and his coworkers[13] produced neurologic disease closely simulating that seen in human pernicious anemia by a diet deficient in pantothenic acid and pyridoxin. Macrocytic hyperchromic anemia did not, however, result.

The treatment of pernicious anemia consists in the continued and persistent use of liver extract, preferably by the injection of a highly concentrated preparation (10 to 15 units per cubic centimeter) given at frequent intervals. The interval varies from patient to patient, particularly according to the degree of neurologic involvement. With well-defined disease of the cord, overtreatment is preferable. Most patients require an injection at least once every two weeks, although some go for a month and others require weekly injections. Relapses occur in those who neglect systematic treatment. The length of time for the development of relapse is a highly individual matter that has been studied by Schwartz and Legere.[14] They suggest that the amount of intrinsic factor secreted by the stomach, the adequacy of the diet, the storage capacity of the liver and certain obscure factors probably influence the duration of the remissions.

A significant problem that seems to be on the increase is the development of hypersensitivity to the injectable liver extract. In some cases, the patient goes into an alarming anaphylactic state, with generalized angioneurotic edema, bronchial asthma, vomiting and diarrhea, drop in blood pressure and

so forth. Schwartz[15] states that allergic reactions occur in about 10 per cent of all patients so treated. In most cases, this is probably due to pork sensitization, since most liver extracts are derived from porcine livers. All that is usually required is to shift to a beef liver extract, such as the 15-unit (1 cc.) Lederle extract or the approximately 1 unit (1 cc.) Campolon (Winthrop) liver extract. Some patients become sensitized to all types of injectable liver, and in these, recourse must be had either to attempted desensitization, which is usually unsatisfactory, or to oral liver extract, oral liver or oral gastric extract (Ventriculin).

Another problem, at least for the pharmaceutical houses, is the matter of assaying liver-extract preparations. This requires the use of relapsed cases of pernicious anemia and the rigid control of such factors as the diet, previous administration of liver or liver extract, daily reticulocyte counts and the use of suboptimal doses of extract. Practically, few relapsed cases suitable for assay are seen, most of the patients having been rendered unsuitable by a previous injection of liver extract or a little liver taken a few days previously. Biologic and animal methods would, of course, be eminently desirable, but thus far no such method has proved of value. Using explants of bone marrow from normal guinea pigs, and measuring the degree of migration of cells in response to control solutions and those containing liver extract, Young and Bett[16] found no consistent results. They were forced to conclude that no difference sufficiently marked to be used as the basis of an assay method existed between the responses to active and inactive extracts.

The greatest problem in therapy is not, however, the use of adequate amounts of properly assayed liver extract in properly diagnosed cases of pernicious anemia, but rather the continued use of liver extract injections in patients who have absolutely no need for this material. In the last year alone, I have personally seen about 20 patients, all of them women, with what I call "Tallqvist anemia" and who have been given continued and persistent injections of liver extract for periods varying from one to twelve years. At some time or other, the patient, her family or a new doctor becomes suspicious about the need for these injections and inquires whether they should be continued. The history is usually that of emotional instability, with numerous spells of feeling "low," "without pep" and so forth. The diagnosis of anemia has been made either by the Tallqvist scale or even more simply by inspection, and the patient is then started on her career of injections. An injection is quickly followed by an improvement in general well-being, but if one or two injections are missed, the patient again feels "low" and the blood is said to "drop." Physical examination in these cases is negative; particular attention should be paid to the tongue and the vibratory sensation, both of which are usually normal. The blood counts are normal, as they might be in a well-treated case of pernicious anemia; the sternal marrow is normal. Gastric analysis is of utmost significance from the standpoint of differential diagnosis; if it shows free hydrochloric acid, as it almost always does, the diagnosis of pernicious anemia may be discarded, together with the necessity for continued injections of liver extract. A curious commentary on this matter is that practically every patient with anemia whom I have seen in the past few years has had numerous injections of liver extract *except those who have had pernicious anemia.* These patients have usually been referred to the hospital because of the possibility of carcinoma or some other serious disease.

Sprue

Both tropical and nontropical sprue are probably to a great extent intestinal disorders characterized by prolonged diarrhea, bulky foul-smelling stools and the development of numerous deficiencies. These probably occur because malabsorption from the bowel results in the deficient absorption of iron, liver-extract principle, vitamin B complex, the fat-soluble vitamins, including vitamin K, and calcium. The disorder is thus fundamentally different from that due to a purely gastric disturbance, such as that seen in pernicious anemia. In sprue, although glossitis and macrocytic anemia may be encountered as in pernicious anemia, there is no evidence of a hemolytic component, the central nervous system is rarely affected, the pigment output in the stool is low, and the slow response to liver extract differs greatly from the quick effect seen in pernicious anemia. Other evidences of intestinal malabsorption include an increased sugar tolerance, a diminished calcium level in the blood, at times associated with tetany, and hypoprothrombinemia. Careful examination of the small intestine by serial roentgenograms and by the "snap-shot" technic revealed changes in 8 successive cases of nontropical sprue characterized by "puddling" and the "moulage sign," together with other indications of a greatly disturbed small-bowel function.[17] Whether these changes are simply secondary to vitamin B-complex deficiency or, as seems more likely, to an intestinal disorder due to multiple factors is not entirely clear at present. Whatever the mechanism, these patients are difficult to treat, often being refractory to liver extract, iron, vitamins and so forth. A crude extract of liver is preferable in view of the multiple deficiencies present. Celiac disease of children is probably a closely related condition.

(*To be continued*)

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 31091

PRESENTATION OF CASE

A thirty-one-year-old lumber contractor was admitted to the hospital because of intermittent hematuria.

About six years prior to admission, without any preceding injury, the patient began to pass grossly bloody urine for periods of two days to two weeks at a time. The periods recurred at intervals, varying from about a week to several months and always cleared up spontaneously. They were usually precipitated by lifting heavy objects. There was no associated pain or tenderness, nor were there any other urinary symptoms.

Two months prior to admission the patient fell, striking the left flank with his elbow. One hour later he again developed hematuria, followed very shortly by severe pain in the left flank. He was admitted to a local hospital, where the hematuria and pain disappeared after several days of bed rest.

X-ray examination of the kidneys at that time revealed extensive calcification, presumably in the pyramids. He was referred to this hospital for study.

For about a year prior to admission the patient had noted blurring of vision, especially when reading. He had also complained of aching in the back and legs for about the same period of time. For seven or eight months preceding entry he had suffered with severe headaches, coming on at any time of the day and usually relieved by aspirin. There was no history of dizziness or tinnitus.

Ten years before entry the patient had had otitis media with mastoiditis, followed by a mastoidectomy. About two years prior to entry he had an attack of uncomplicated "scarlet fever," but continued to work throughout his illness.

Physical examination was essentially negative. The temperature was 98.6°F., the pulse 62, and the respirations 20. The blood pressure was 145 systolic, 85 diastolic.

Examination of the blood revealed a red-cell count of 4,830,000, with 15.4 gm. of hemoglobin, and a white-cell count of 7600, with 61 per cent neutrophils. Repeated urine examinations revealed a specific gravity ranging from 1.004 to 1.010, with + to +++ tests for albumin. The Sulkowitch test for urinary calcium phosphate was + to ++. The sediment (catheterized specimen) contained innumerable white cells and an occasional red cell and granular cast. Urine cultures showed moderate numbers of colon bacilli and nonhemolytic streptococci on a few occasions and no growth on others. The serum nonprotein nitrogen was 20 mg. per 100 cc., the protein 5.7 gm., the chloride 98 milliequiv. per liter and the carbon dioxide combining power 28.2 millimols. Several blood calcium determinations ranged from 11.1 to 11.6 mg. per 100 cc.; the phosphorus was 2.7 to 3.0 mg., and the phosphatase 5.5 Bodansky units. A phenolsulfalein test revealed 90 per cent dye excretion in one hour. The blood Hinton test was negative.

An intravenous pyelogram revealed multiple fine dense irregular areas of calcification having the general arrangement of staghorn calculi in both kidneys, more marked on the right. There were no areas of calcification in the ureters or bladder. The intravenous dye was excreted promptly. The pelves were of normal size. The calyxes on the right were obscured by calcification, but those on the left did not appear dilated. The upper end of the left ureter was moderately dilated. The bones were of normal density, and no cysts were seen.

A retrograde pyelogram on the fourth hospital day revealed that the areas of calcification described were within the kidney pyramids and bulged into the calyxes, especially into the upper calyxes on the left and the lower ones on the right. The cupping of the minor calyxes was markedly increased, but there was no appreciable dilatation. The ureters were dilated down to the bladder and were moderately tortuous.

*On leave of absence.

On the eleventh hospital day an operation was performed.

Differential Diagnosis

Dr. Edward C. Reifenstein, Jr.: This discussion can be divided into two parts — the nature of the lesions in the urinary tract, and the cause of these lesions.

The pathology in the urinary tract first indicated its presence by intermittent gross hematuria. The most frequent causes of grossly bloody urine are tumors, calculi and tuberculous infection of the urinary tract. The onset of hematuria with exertion favors a tumor or a calculus; the persistence of intermittent bleeding for over five years without other symptoms is rather against a tumor or tuberculous infection. The recent episode of pain in the left flank following trauma and the aching in the back and legs for about one year are consistent with calculi; the findings on x-ray examination of the urinary tract confirm the diagnosis. The appearance on the x-ray plates is that of small deposits of calcium in the tubules of the pyramids of both kidneys of the type called "nephrocalcinosis." I do not believe that this type of calcification has been observed in tuberculous disease of the kidney, and hence I shall not give further consideration to an acid-fast infection in the kidney. We shall assume that the hematuria is due to the nephrocalcinosis.

No areas of calcification were demonstrated in the ureters or bladder, and the pelves were of normal size. Both ureters were dilated and tortuous, findings that are rather hard to explain. There was no history of ureteral obstruction during the passage of stones, although it is conceivable that one may have been passed during the episode of left flank pain. This would not explain dilatation of the right ureter. It is also possible that congenital strictures may have been present. Dilatation of the ureters, however, does occur for reasons that are not entirely clear in connection with renal infection, and presumably this is the explanation in the case under discussion. Furthermore, the passage of blood clots would aggravate the dilatation.

It is clear that this patient had pyelonephritis: the fixation of the specific gravity of the urine, the albuminuria and the finding of innumerable white cells, of occasional red cells and granular casts and of colon bacilli and nonhemolytic streptococci are evidence of this. The satisfactory excretion of phenolsulfonephthalein dye and of the intravenous dye for the pyelogram and the relatively normal serum nonprotein nitrogen level indicate the lack of glomerular involvement. As a matter of fact, the serum nonprotein nitrogen of 20 mg. per 100 cc. is slightly above the average normal value of 15 mg., and is consistent with the chloride level of 98 milliequiv. per liter, which is on the low side of average normal (103 milliequiv.), and with the carbon dioxide combining power of 28.2 millimols, which is slightly on the high side of average normal (25 millimols). These slight changes are probably indicative of a mild state of dehydration. They are of no importance except to indicate that the low serum protein level (5.7 gm.) is even more significant than is apparent from the comparison with the normal levels of 6.5 to 8 gm. The hypoproteinemia was presumably due to the proteinuria.

Before I discuss the cause of these lesions, I shall consider for a moment the chemical nature of the calcification in the kidneys. Nephrocalcinosis of the type present in this patient is usually due to deposition of calcium phosphate concretions in the tubules. It is probable that the calcification increased after the pyelonephritis was established. This additional growth of the calculi would also tend to be deposition of calcium, since calcium tends to be thrown out in an alkaline urine, and colon bacilli and nonhemolytic streptococci are known to split urea and form an alkaline urine.

The second part of the discussion has to do with the cause of the nephrocalcinosis and of the pyelonephritis. The first question to decide is whether the pyelonephritis preceded or followed the nephrocalcinosis. Both situations are possible. There is little evidence, however, that the pyelonephritis preceded the calculi in this patient. We have assumed that the hematuria was due to the calculi. There was no history of renal disease prior to the hematuria. Although the patient did have otitis media and mastoiditis four years before the onset of hematuria, no renal complications are noted as sequelae to these infections. It is perhaps significant, therefore, that four years after the onset of hematuria the patient had an attack of what is called "uncomplicated scarlet fever," throughout which he worked. This may have been the infection that led to the pyelonephritis, which could readily have been established in kidneys already damaged with calcification. The case report makes no reference to the status of the teeth. Examination of the x-ray films of the jaws reveals at least two apical abscesses. Since spread of infection from an apical abscess to the kidney is quite frequent, the pyelonephritis may have had its origin from the infection around the teeth. We have no way of knowing how long these abscesses had been present. It is of interest, however, that only a year after the attack of scarlet fever the patient began to have blurring vision and severe headaches, both of which could be manifestations of renal infection. These symptoms were present only about one year before admission. Some significance might be given to the systolic blood-pressure level, which was perhaps a little higher than one would expect unless the patient was beginning to develop hypertension. The finding is consistent with a pyelonephritis of rather short duration. Further evidence that the nephrocalcinosis was present before the renal infection is derived from the fact that when pyelonephritis causes cal-

culus formation in the kidney it produces an entirely different type of calcification, which is in the renal pelvis rather than in the pyramids themselves.

If we assume that the nephrocalcinosis preceded the pyelonephritis, we then have to determine why the patient developed the nephrocalcinosis. Calcium deposits in the kidney occur from a supersaturation of calcium in the urine. In the absence of infection, in general, there are four causes for such a supersaturation: ingestion of too much calcium, leading to hypercalciuria; ingestion of too much alkali, leading to an alkaline urine, which favors calcium deposition; a blood acidosis, leading to resorption from bone of excessive amounts of calcium and to excretion of excessive amounts of calcium as a fixed base to get rid of acid radicles; and excessive parathyroid hormone activity, leading to phosphorus diuresis, a low serum phosphorus level, undersaturation of the blood with respect to calcium and phosphorus, increased resorption of calcium and phosphorus from bone and, finally, hypercalciuria. Since there is no history in this case of polygalactodipsia (increase in milk intake), alkalophagia or acidosis, the first three of these causes will be dismissed.

Did the patient have hyperparathyroidism? If so, there was practically no bone disease, since the visible bones are of normal density and show no cysts, the lamina dura about the teeth is fairly well preserved, and the serum phosphatase level was practically normal (normal, 3 to 5 Bodansky units per 100 cc.), particularly if the slight dehydration is recalled. It has been well established by Dr. Fuller Albright, however, that hyperparathyroidism can exist with little or no bone involvement, presumably because the patient ingests sufficient calcium and phosphorus to maintain the blood saturation so that bone resorption does not occur. The serum calcium and phosphorus levels make a diagnosis of hyperparathyroidism attractive. The calcium levels of 11.1 to 11.6 mg. per 100 cc. are in the borderline range between normal and high values according to the laboratory methods in use before the fall of 1943 (normal, 9.0 to 10.5 mg.) and are definitely high according to the methods now in use (normal, 8.5 to 10.0 mg.). This patient was studied in 1942, so the serum calcium level must be judged by the old standard. When these values are corrected for the low serum protein level, they would average about 12.0 mg. or a little more; in other words, they would be unquestionably high. Furthermore, the serum phosphorus values were definitely low (normal, 3.0 to 4.0 mg. per 100 cc.), particularly if we recall that slight dehydration was present. The low serum phosphorus levels also make it clear that the overactivity of the parathyroid gland was primary and not secondary to renal disease, for in the latter situation the serum phosphorus level is high.

Further strong evidence in favor of hyperparathyroidism is the occurrence of nephrocalcinosis. This type of calcification in both kidneys rarely occurs except as a consequence of hyperparathyroidism. A somewhat similar picture, but with larger areas of calcification, has been observed in 2 cases with *Haemophilus influenzae* infection in the urine[1]; this condition is quite rare, and there is nothing to suggest it in this patient. Other conditions with calcification in the pyramids, such as mercury poisoning, late rickets and focal deposits due to colon-bacillus infection, are also extremely rare; furthermore, they do not give the finely granular calcification seen in the films of the present case. The fact that the urinary calcium excretion was not abnormally high, as measured by the Sulkowitch test, is consistent with the diagnosis, since the specific gravity of the urine is fixed in a low range. It would be of interest in obtaining further support for the diagnosis of hyperparathyroidism to know whether the patient had complained of fatigue, polydipsia or polyuria, and whether he had lost weight.

In summary, the sequence of events seems to have been: hyperparathyroidism, probably due to a small parathyroid adenoma; nephrocalcinosis, with calcium phosphate deposits; and pyelonephritis possibly due to colon bacilli and nonhemolytic streptococci as a result of the attack of scarlet fever or of spread from the apical abscesses.

A brief word about treatment is in order. As a first step the parathyroid adenoma should be removed. Then efforts should be made to improve the kidney status. This type of calcification is not amenable to surgery, but retrograde dissolution of part of the calcification might be possible with Solution M (magnesium citrate solution buffered at a pH of 5).[2] There is still hope that an agent may be found that, taken by mouth, will tend to dissolve the calcium by creating an acid situation in the tubules. Of course, the urinary-tract infection must be kept under control during such a procedure. Even if no attempt is made to get rid of the calcification, an effort should be made to eliminate the urinary infection. Although this is difficult to do in the presence of calculi, it has been accomplished in several cases. In particular, this patient might have been one of those in whom penicillin causes a disappearance of nonhemolytic streptococci, and then colon bacilli, which occasionally are secondary invaders, may disappear spontaneously or in response to sulfonamide therapy.

DR. BENJAMIN CASTLEMAN: Dr. Colby, would you like to comment on this case?

DR. FLETCHER H. COLBY: We occasionally see patients like this, with calcification spread throughout the kidney in such a way that it involves the parenchyma rather than the calyxes and pelves. I recently had a patient, a young man in the air

force, who presented a picture almost exactly the same as this. He had calcification spread throughout the parenchyma of both kidneys. The urine was strongly acid, and he had a colon-bacillus infection.

I was interested in Dr. Reifenstein's comments on the distribution of calcification in tuberculosis. My experience has been that calcification in tuberculosis is present anywhere in the kidney. When we see patients like this we always think that tuberculosis is one thing that we have to eliminate. I have always hesitated to eliminate tuberculosis simply on the x-ray appearance of the distribution of calcium. I may be entirely wrong, but I believe that one ought to depend entirely on guinea-pig inoculation to be sure that it is not tuberculosis, even though the patient has an accompanying streptococcal or colon-bacillus infection, which is frequent in tuberculosis.

It seems to me that we do not know the answer to this type of case, whether the disease starts as pyelitis and, as a result of long-continued renal damage, calcium is deposited or whether the calcium is deposited first as a result of the infection.

Dr. Fuller Albright: I did not know that tuberculosis would cause bilateral calcification so uniformly throughout the kidney. It is usually bilateral but not so smoothly spread.

Dr. Oliver Cope: As one would expect from what Dr. Reifenstein has said, exploration for an anatomic diagnosis was an inevitable requirement. We can sit and argue at this stage about the diagnosis but we need more positive evidence. Early operation is important from the patient's point of view because some of our best results in hyperparathyroidism have been achieved in patients who have had a mild degree of glandular disease with crippling kidney damage. The amount of kidney damage may be wholly disproportionate to the glandular dysfunction.

The surgeon likes to be asked to operate on patients with an unequivocally high blood calcium level, an unmistakably low phosphorus level and a great deal of calcium in the urine. In hyperparathyroidism, as Dr. Castleman[3] has pointed out, the size of the offending parathyroid adenoma is directly proportionate to the increased level of ionized calcium in the blood plasma; in a patient with a blood calcium level of 14 mg. per 100 cc. or higher one knows that the surgical problem is simple because the offending gland or glands will be at least grape size and easy to identify. The surgeon approaches a patient of this type, one having a blood calcium level just above normal, with his heart in his mouth, and as you will see in this patient I was not able altogether to swallow my heart when I had finished. I shall read part of the operative note.

> The right thyroid region was first explored. A small, thin, soft gland, the right upper, was found in a sulcus of the thyroid gland posteriorly, at the junction of the middle and upper thirds. It was the brown color of an adenoma but was too small and thin. [The color of the gland depends on the proportion of fat to epithelial cells. If it contains no fat, it is brown; the more fat it contains, obviously the yellower it is. Normal glands vary. Adenomas generally have no fat in them.] The left side was then explored. Both the upper and lower were identified. The upper was asymmetrically placed, near the inferior thyroid artery. It was of the same brown color as the right upper but was larger and firmer. The left lower was grayer in color, longer and a little softer and lay posterior to the lower pole. The lower right region was next explored, and the gland was found. It was the largest, but was the gray-greenish color of the left lower. The right lower was excised, since it was thought to be an adenoma, but after removal it seemed more like a normal gland. The left upper was then excised as the likeliest to be an adenoma, the left lower and right upper remaining intact and unbiopsied.

Thus we found the four glands; the two upper ones were brown, and the two lower were a totally different color, the color of the largest gland not being consistent with adenoma. Within the normal-sized glands in such mild cases one can often find an adenoma that does not appear on the surface. Seemingly that is what we were confronted with here.

Clinical Diagnoses

Parathyroid adenoma.
Hyperparathyroidism.

Dr. Reifenstein's Diagnoses

Hyperparathyroidism (parathyroid adenoma).
Nephrocalcinosis (calcium phosphate stones).
Pyelonephritis (colon bacilli and nonhemolytic streptococci).

Anatomical Diagnosis

Parathyroid adenoma (parathyroidoma).

Pathological Discussion

Dr. Castleman: The gland that we received was quite small, and the size of the upper limit of a normal gland, and triangular. On microscopic examination, however, there was no doubt that it was an adenoma. There was a narrow rim of normal parathyroid chief cells and numerous fat cells surrounding the tumor, which had no fat cells and was composed of large water-clear cells. This type of tumor should probably be called not an adenoma but a parathyroidoma, because the cells do not form glands. This is one of the less frequent varieties of tumor that we see in this disease. The water-clear cells are enlarged vacuolated chief cells. In the usual type of adenoma, the chief cells are slightly enlarged and uniform in size and have a pale acidophilic cytoplasm. Another type is one that shows a bit more variation in the chromatin of the cells and sometimes has giant nuclei. This is the type that has been referred to by some pathologists as a parathyroid carcinoma. In our experience covering more than eighty tumors we have been unable to find a single example of parathyroid carcinoma. There have been no recurrences or evidence of metastases.

In a recent paper from the Mayo Clinic reporting 14 cases of hyperparathyroidism, 13, or 92.8 per cent, are called carcinomas.[4] I believe that this paper gives the public the wrong impression of hyperparathyroidism, because this condition is benign, not malignant. Their interpretation is a bit weakened by a statement at the end, "In no case in the series under study did metastases or local recurrence occur." In other words, the diagnosis was wholly based on the morphology of the cell. The evidence for the malignant character of these tumors, according to the Mayo Clinic pathologists, rests on hyperchromatism and irregularity of the size of the nuclei. They show no definite mitoses in their photomicrographs. We have seen this same irregularity in staining character and in the size of the nuclei but do not believe that this justifies tagging a patient with carcinoma. In addition, the benignity of the disease is well borne out by the results.

A true carcinoma of the parathyroid gland can occur. Most of these, however, are nonfunctioning. In the Mayo Clinic report, reference is made to the fact that a review of the literature disclosed 7 cases of functioning parathyroid carcinomas. Two of these were from the Mayo Clinic and were included in the series of 14. This leaves only 5 cases outside of the Mayo Clinic. Some time ago Dr. Cope and I reviewed the reports of these 5 cases and concluded that only 2 of them[5,6] were definitely malignant. It would be extremely unusual, therefore, to find carcinoma in 92.8 per cent of one series — that of the Mayo Clinic — and only 5 cases, less than 2 per cent, in the rest of the 300 reported cases. I have dealt with this aspect of the disease because I do not want you to go away with the idea that hyperparathyroidism is caused by a parathyroid carcinoma; it should still be considered a benign condition.

DR. FULLER ALBRIGHT: This case is an example of a tiny tumor, no bigger than a normal gland, that caused hyperparathyroidism. I think the point is that a normal gland is three quarters fat and one quarter epithelial cells, whereas a tumor is entirely composed of epithelial cells.

DR. CASTLEMAN: I might add that I have received a letter from this patient's physician stating that in November, 1944, more than two years after operation, the patient's general health was excellent and that he was regularly doing heavy work. It is interesting, however, that the urine was cloudy and that x-ray films still showed calcific deposits in the tubules.

DR. COPE: I am glad that Dr. Castleman has dealt with the question of carcinoma in these parathyroid tumors. I am astounded at the position taken by the physicians of the Mayo Clinic. They are calling the common benign adenoma malignant. The surgical implications that they draw are inordinate. Widespread excision, which one likes to practice in malignant disease, would be crippling to the patient.

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CASE 31092

PRESENTATION OF CASE

First admission. A thirty-four-year-old accountant was admitted to the hospital after a convulsion.

Sixteen years previously, at the age of eighteen, and many times since then, he had been refused life insurance because examination of urine revealed albumin, casts and a low specific gravity. At that time eight different physicians saw him in rapid succession and treated him with "bladder irrigations." At the age of twenty he was admitted for two weeks to another hospital, where he was cystoscoped, following which he had weekly bladder irrigations for one year. At twenty-seven he was placed on a salt-free, low-protein diet, which he disregarded. At twenty-nine he was admitted for two weeks to still another hospital, where he was given sulfathiazole, which "cleared up" the urinary-tract infection. He was apparently well for the next five years. Five months before admission he experienced severe muscular cramps in the calves and upper abdomen. At that time his physician gave him calcium lactate and iron tablets and three months later added one quart of milk daily. Two weeks before admission he developed a head cold with coryza, severe hoarseness and cough productive of bloody mucus. He spent three days in bed, the oral temperature being 102°F. While in bed his muscle cramps returned. Two days before admission he experienced two episodes of shaking of the extremities and body without loss of consciousness but with inability to grasp anything and with resultant pain and tenderness of the muscles. In the afternoon of the day of admission, while standing at a gasoline station, he had another episode of generalized shaking and fell to the ground unconscious, following which he was brought to this hospital.

At fourteen years of age he had had a tonsillectomy, and at eighteen he had been treated for gonorrhea with irrigations. He had been married twice and had two children, six and nine years of age respectively.

Physical examination revealed a well-developed, well-nourished, man showing deep regular respirations. The throat was red. The right side of

the tongue was swollen and bruised. The nasal mucosa was covered with dried hemorrhagic exudate. A few petechiae were present over the upper thorax. Breath sounds were slightly increased, with fine rales in both axillas. An apical Grade II systolic murmur was heard. The muscles were diffusely tender. A uremic odor was noted on the breath. Trousseau and Chvostek signs were negative.

The temperature was 98.6°F., the pulse 90, and the respirations 20. The blood pressure was 160 systolic, 80 diastolic.

Examination of the blood showed a red-cell count of 2,380,000, with 6.6 gm. of hemoglobin. The white-cell count was 14,700, with 77 per cent neutrophils. There was moderate variation in size and shape of the red cells. The urine was light amber and acid in reaction. The specific gravity ranged from 1.008 to 1.012, with one later specimen reaching 1.020 and one 1.004. There was a ++ test for albumin. The sediment showed 1 to 3 white cells and occasional red cells per high-power field. A Hinton test was negative. The nonprotein nitrogen was 120 mg. per 100 cc. The total protein was 5.5 gm., with an albumin-globulin ratio of 2.5. The serum sodium was 131 milliequiv. per liter, the chloride 106 milliequiv., the carbon dioxide 7 millimols, the calcium 8.6 mg. per 100 cc., the phosphorus 7.5 mg., the potassium 5.7 milliequiv. per liter, and the phosphatase 4.9 Bodansky units per 100 cc. A phenolsulfonephthalein test showed less than 10 per cent excretion in one hour. Urine culture on admission grew a few colonies of *Staphylococcus albus*. X-ray films of the abdomen were negative.

On the twelfth day the patient was put on intramuscular penicillin — 48,000 units per day for four days, 24,000 units for two days and 12,000 units for six days. Subsequent urine cultures showed no growth. On the nineteenth day he had had a severe pain in the chest, which one observer believed was characteristic of coronary occlusion. An electrocardiogram was negative. During treatment the carbon dioxide reached 31.2 milliequiv. per liter but fell back to 17 milliequiv. The serum phosphorus was elevated to 11.4 mg. per 100 cc. The nonprotein nitrogen did not change.

The patient was discharged on the thirty-fifth hospital day; he felt quite strong and much improved. During his stay he had received six 500-cc. transfusions of whole blood, as well as Citrocarbonate and calcium gluconate.

Second admission (two months later). Six weeks after discharge he had an episode of severe nocturnal dyspnea. He promptly developed congestive failure but responded well to digitalization. Ten days before admission he developed a cold, with fever and a cough productive of blood-tinged sputum, and a week later had another episode of severe dyspnea. During the following two days he became increasingly drowsy, continued to cough, was nauseated, vomited and had slight pain in the right chest.

Physical examination revealed a pale, dehydrated, drowsy, orthopneic man sitting upright in obvious distress; he had a strong uremic odor on his breath. The heart was enlarged in all directions. The sounds were weak, with a gallop rhythm at 120. The lower two thirds of the right chest showed dullness and diminished breath sounds and tactile fremitus. Moist rales were heard bilaterally. The pain in the right chest was severe. The liver was palpable three fingerbreadths below the costal margin. There was moderate ascites, and marked edema of the sacrum and lower extremities.

The temperature was 101°F., and the respirations varied between 20 and 35. The blood pressure was 210 systolic, 100 diastolic.

Several attempts to withdraw fluid from the right chest failed. A pericardial friction rub was constantly present after the third day. The urine was alkaline, with a specific gravity of 1.007, a + test for albumin and a few red cells and white cells in the sediment. The blood revealed a white-cell count of 44,200, with 99 per cent neutrophils, and a red-cell count of 1,840,000, with 5 gm. of hemoglobin. The nonprotein nitrogen was 200 mg. per 100 cc., the carbon dioxide, 12 to 15 millimols per liter, the chloride 91.0 milliequiv., the calcium 8.1 mg. per 100 cc., and the phosphorus 6.5 mg. Urine culture grew *Staph. albus* and nonhemolytic streptococci. The blood and sputum showed Type 1 pneumococci.

The patient was treated with intravenous fluids, sulfadiazine, transfusions, Mercupurin, digitalis, calcium gluconate, Hykinone and sedative drugs. The sulfadiazine blood level never exceeded 10 mg. per 100 cc., and the urinary output did not drop. His sensorium gradually became increasingly clouded. He was comatose and died on the eighth hospital day.

Differential Diagnosis

Dr. William W. Beckman: It seems to me that three diseases contributed to this man's death. One of them was acute and terminal. Another, hypertensive heart disease with congestive failure, was a sequela of the third, chronic renal disease. Certainly there is enough evidence to make a diagnosis of chronic nephritis of some sort. He had a long history of continuous albuminuria, low specific gravity and casts in the urine. He evidently was having symptoms during most of this period because he consulted so many physicians.

On physical examination there was the additional finding of high blood pressure. The laboratory findings of marked anemia, albuminuria, elevated nonprotein nitrogen, elevated serum phosphorus and marked acidosis all confirm the diagnosis of chronic nephritis. It is of interest that the casts had disappeared from the urine by the time he came to this hospital. This of course is to be expected, for, as chronic nephritis progresses into the terminal stage,

the formed elements in the sediment usually diminish and frequently disappear.

One must always attempt to define the kind of nephritis. This differential is usually more academic than practical, but it has gained importance as various sorts of chemotherapy have been devised to treat pyelonephritis according to the type of infecting organism. The wide range of the specific gravity, from 1.004 to 1.020, is surprising, for one would expect a fixed specific gravity in the terminal stage of the disease. No correction was made for the albumin, however, and it is well known that unless such a correction is made the specific-gravity readings may vary markedly. There is little here to suggest glomerulonephritis. He had had a tonsillectomy, but he had had no evidence of a streptococcal pharyngitis, scarlet fever or anything that tends to dispose toward the development of glomerulonephritis. Everything seems to point to the fact that he had pyelonephritis. It would be interesting to know whether he consulted a physician at the age of eighteen for gonorrhea before he was refused life insurance, owing to albuminuria. If this had been the case it is possible that the local irritation occasioned by the irrigations might have started an infectious process in the genitourinary tract. Following the onset, whatever it was, his many physicians all seemed to think that he needed the various sorts of treatment for the urinary-tract infection that were used. He was given bladder irrigations before the time of chemotherapy, and when chemotherapy came along, he was given sulfadiazine, which was said to clear up the infection. Probably it did not. He did well following this form of treatment and had five years of freedom from symptoms. This was probably because he had become accustomed to his chronic nephritis rather than because it had been cured, although there is no way of proving that. At any rate, I think that pyelonephritis is the likeliest explanation for the chronic renal disease.

The fact that he had pains in the legs and abdomen and had an attack of pain in the chest, which was thought to be characteristic of myocardial infarction, and the fact that he had cerebral manifestations in the form of convulsions make me wonder about some generalized vascular disease, such as periarteritis nodosa. Certainly all these are symptoms of that syndrome, and in addition it would explain the petechial rash that he had at the time of the first admission. Otherwise I cannot explain the rash. One does get all kinds of skin manifestations in periarteritis nodosa. I think it unlikely, however, that a person with periarteritis nodosa could go for sixteen years with only albuminuria, and little else, to show for it. We are following one patient with periarteritis nodosa, proved by biopsy, of many years' duration who shows nothing but albuminuria. That is most unusual. Anyway, if a patient has chronic pyelonephritis he has a good right to a general vascular disease in the form of plain arteriosclerosis, and I should think that that is the more probable in this case.

It is worth considering the nature of the convulsions. In uremia there are two varieties — those due to hypertensive encephalopathy and those due to tetany resulting from a low serum calcium. The low calcium in this case is probably associated both with a lowered serum protein and with definite phosphate retention. Incidentally in situations like this, when there is chronic phosphate retention, the parathyroid glands are almost invariably found to be hypertrophied. This has been interpreted to mean that these glands are working overtime in an effort to correct the derangement in calcium and phosphorus in the serum. A total calcium of 8.6 mg. per 100 cc. is hardly low enough to produce tetany, particularly in the presence of such a high degree of acidosis as is indicated by the carbon dioxide of 7 millimols per liter. Therefore, I should say that the convulsions were on the basis of hypertensive encephalopathy, although I do not know what that is.

We then come to the treatment with penicillin. That is something not too commonly done in uremia. I have a similar patient, a man with chronic uremia whom I have followed for seven years. His urine culture always grows *Staphylococcus albus*. I asked Dr. Forbes about treating him with penicillin, and she replied that it would certainly be an advisable thing to do. It might be well to have Dr. Forbes comment on that now.

Dr. Anne P. Forbes: My statement was based on the fact that we know that a chronic renal infection can lower kidney function. Since these patients have little renal tissue to spare, getting rid of the infection may relieve them of a burden that might otherwise be crippling.

Dr. Beckman: Whether it was due to penicillin, — he received a number of other things, — the patient improved and went home feeling better. It is interesting that they treated him with alkalies in the presence of the low calcium. Alkalosis, of course, tends to accentuate any tendency to tetany. I suppose that the addition of calcium gluconate was meant to take care of this eventuality.

The attack of nocturnal dyspnea indicates that the patient had developed hypertensive heart disease and congestive failure. Although his failure improved somewhat at first it was quite definitely manifest when he came into the hospital the second time.

The final episode clearly appears to be a Type 1 pneumococcus infection of the lungs and blood stream. It is not easy to take care of such a pneumococcal infection in the presence of chronic renal disease. In addition to the therapeutic problem presented by the necessity of giving chemotherapy and fluids to a person with chronic renal disease

who is in congestive failure, another dilemma is encountered. The chronic renal disease makes it much more difficult for the body to take care of the infection, and at the same time the acute infection tends to accentuate the renal failure.

I think that this patient died of Type 1 pneumococcus infection, both in the lungs and in the blood stream, of chronic pyelonephritis, with renal failure, and of hypertensive heart disease, with severe congestive failure. I should have pointed out that I believe the large liver and the ascites were on the basis of congestive failure and that the pericardial friction rub indicates a terminal uremic pericarditis.

DR. JACOB LERMAN: While the patient was being treated in the hospital, we talked a bit about the cause of the tetany and I thought definitely when the patient was first admitted that the tetany was due to overbreathing. I do not believe we settled that point. In any case the patient got better and was treated with alkalies and calcium and the convulsions did not return.

CLINICAL DIAGNOSIS

Chronic pyelonephritis.
Lobar pneumonia, Type 1.

DR. BECKMAN'S DIAGNOSES

Chronic pyelonephritis.
Uremia.
Pneumonia and septicemia (Type 1 pneumococcus).
Hypertensive heart disease.
Pericarditis, terminal, uremic.
Congestive heart failure.

ANATOMICAL DIAGNOSES

Chronic pyelonephritis, bilateral.
(Uremia.)
Pleuritis, pericarditis and peritonitis, acute fibrinous, with effusion (Type 1 pneumococcus).
Septicemia (Type 1 pneumococcus).
Parathyroid hyperplasia, marked, secondary.
Cardiac hypertrophy, hypertensive type.
Peripheral edema.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: The autopsy on this man showed that the terminal infection was a Type 1 pneumococcus septicemia, with a shaggy fibrinous pericarditis, pleuritis and peritonitis, in other words, an acute fibrinous serositis. I do not believe that I have ever seen all three serous cavities affected by the pneumococcus before. I suppose that the infection started in the lung, although at the time of autopsy we were unable to find anything definite in the lung. If there had been pneumonia, it had resolved. The parenchyma just under the pleura showed slight chronic pneumonitis, which may have resulted from the pleurisy, although it is quite possible that there had been a localized pneumonia. The kidneys were quite small, one weighing 50 gm., and the other 90 gm. They were markedly contracted and pale and showed the characteristic picture of pyelonephritis. There was marked destruction of the tubules and glomeruli and extensive infiltration with lymphocytes and polymorphonuclears. There were tubular atrophy and compensatory hypertrophy.

Long-standing chronic renal disease frequently produces secondary hyperplasia of the parathyroid glands, and in this case all four were tremendously enlarged, weighing together almost 5 gm., which is about thirty to forty times their normal weight. They were pale gray in contrast to the brown color that one sees in primary parathyroid adenoma. Microscopically there was an increase in the number of normal-sized cells rather than the hypertrophy of individual cells that is seen in an adenoma, with obliteration and replacement of all the fat cells.

The heart was enlarged, weighing over 500 gm.

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SEVENTY-FIFTH ANNIVERSARY

It is not every day that a state department of health can celebrate a diamond jubilee. In fact, it has never happened in America until now. This issue of the *Journal* carries an article outlining the history of the Massachusetts Department of Public Health during the past seventy-five years, stressing in particular the contributions made by certain outstanding medical men.

At the time of the founding of the Massachusetts State Board of Health in 1869, the term "public health" meant very little in this country. Measures for the control of communicable disease were based on beliefs rather than on knowledge. Robert Koch and Louis Pasteur had not yet revolutionized the prevailing ideas on the causation of disease. Only in the case of smallpox was there an effective method of immunization. Public water supplies were far from safe. Sewage disposal was still largely a matter of individual rather than community responsibility.

The newly created State Board of Health attacked these problems with courage and wisdom. They pioneered so broadly and so successfully that a lasting impress was left on sanitary practice in this and even in other countries. This was made possible largely because of a group of extraordinary individuals, who devoted their best energies to the task. Chief among these early pioneers were Dr. Henry I. Bowditch, Dr. Henry P. Walcott and Dr. Theobold Smith, honored names in Massachusetts medical history.

The period from 1869 to 1914 has well been called the "era of sanitation." By the end of that period, tremendous strides had been taken through the energetic programs of Mills, Sedgwick, Goodnough and other engineers and the time was ripe for entry into new fields. When the board was succeeded by the Department of Health in 1914, which became the Department of Public Health in 1919, an expansion in the perspective of the public was taking place and new tasks were undertaken.

Emphasis in the control of communicable diseases shifted to prevention through the use of biologic products. Education of the public in matters of hygiene was recognized as a necessity. Dr. Allan J. McLaughlin, Dr. Eugene R. Kelley, Dr. George Bigelow and their successors, following in the footsteps of the original board, kept the department well up in the front line of advance in public-health knowledge and practice. Indeed, on various occasions the department led the advance, as in the cancer-control program and that for the study and control of tuberculosis in children. A constant stream of visitors from all over the world to observe the workings of an established state health organization has testified to the department's standing.

It is especially interesting to study the relation of state to local administration in Massachusetts in view of the strong home-rule tendencies of this state. The department's functions have been mainly advisory, rather than supervisory, from the beginning.

Its seventy-five years' experience furnishes material for a study of decentralized health administration, as contrasted with the more centralized forms of many other states. Some of the inadequacies of the decentralized system are apparently now coming to light as problems become more complicated because of the widening scope of medicine and public health and the increasing density of population. An arrangement that functioned satisfactorily in pioneering days may prove inadequate to cope with the problems of the future.

MATERIA MUSICA

THE Massachusetts General Hospital in 1941 opened the doors of its new chapel, the creation of which was the "postscript" of Bishop Lawrence's long and useful life. A Hammond organ became part of the equipment of this nonsectarian sanctuary, and to it has been added an electrical record player of high quality. With also a portable instrument that can be taken to the wards, the hospital has at last, within this decade, achieved musical independence as a long step forward from the time when hired or volunteer talent was brought to its corridors on infrequent occasions.

A program is now in effect whereby regular organ recitals and occasional gatherings of whoever will for singing exercises are augmented by short concerts of electrically transcribed music at established hours each week. In this way is the chapel, although used for no set services, still further fulfilling the broad purposes of its founder.

The next step in the musical education of the institution has been, and will continue to be, the collection of a library of records — an enterprise that has gone forward surprisingly well as its needs were made known. Although now under the guidance of a music committee, this new activity is in the hands of the Occupational Therapy Department — fittingly enough, if it is conceded that the minds and emotions should be as well occupied as the hands.

No one will question the therapeutic value of music or the possibilities for its use in the hospital armamentarium that the future may bring. In the meantime the Massachusetts General Hospital may be congratulated on the opportunities that it has been able to make available for patients and personnel, with our best wishes for continuing success in this cultural project.

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

BISHOP — William A. Bishop, M.D., of Watertown, died February 14. He was in his fifty-seventh year.

Dr. Bishop received his degree from Tufts College Medical School in 1913. He was a widely known authority in the field of industrial medicine, and for the past fifteen years had served as medical director for the American Mutual Liability Insurance Company. During World War I, Dr. Bishop served as a captain in the Army Medical Corps. He gave up his private practice last October but had continued as associate physician on the staff of the Massachusetts General Hospital. He was a fellow of the American Medical Association.

His widow and a daughter survive.

SHAUGHNESSY — M. James Shaughnessy, M.D., of Framingham, died February 17. He was in his sixty-fourth year.

Dr. Shaughnessy received his degree from Harvard Medical School in 1907.

His widow survives.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

EMERGENCY MATERNITY AND INFANT CARE PROGRAM

CONSULTANTS IN PEDIATRICS

There has been a change in the procedure concerning payment to pediatric consultants participating in the EMIC program.

The department is no longer permitted to pay a consultant's fee to a pediatric consultant when he signs the pediatric application, because he then becomes the attending physician and must be paid as attending physician according to the fee schedule. A pediatric consultant is considered to be the attending physician when the case is turned over to him for care by another physician or when the mother goes directly to him for the care of her infant.

A consultant in pediatrics who expects to be paid as a consultant should make sure that the pediatric application is signed by the attending physician and that he names the pediatric consultant on the application. The pediatrician may then be paid as a consultant to the attending physician when services are rendered.

CO-OPERATION REQUESTED IN BLINDNESS PROGRAM

Attention is called to the law requiring the reporting of blindness in any patient examined at a clinic,

hospital or other institution or by a private physician or optometrist.

Such information is indispensable as a foundation for the development of a sound constructive program for preventing blindness in Massachusetts. As complete knowledge of the causes of blindness is vitally necessary, the co-operation of all physicians is urged in observing the law, which is as follows:

Chapter 69, General Laws, Section 19A: Whenever, upon examination at a clinic, hospital or other institution, or elsewhere, by a physician or optometrist, the visual acuity of any person is found to be with correction 20/200 or less in the better eye, or the peripheral field of his vision to have contracted to the ten degree radius or less regardless of visual acuity, the superintendent of such institution, or the physician, optometrist or other person who conducted or was in charge of the examination if it took place elsewhere than in such an institution, shall within thirty days report to the director the result of the examination and that blindness of the person examined has been established.

Such information should be forwarded to: Arthur F. Sullivan, Director, Division of the Blind, Department of Education, 110 Tremont Street, Boston 8. A supply of report forms may be obtained from the same address.

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR JANUARY, 1945

Résumé

Diseases	January 1945	January 1944	Seven-Year Median
Anterior poliomyelitis	2	1	1
Chancroid	3	0	*
Chicken pox	1798	2512	2528
Diphtheria	20	24	17
Dog bite	594	460	511
Dysentery, bacillary	43	23	6
German measles	156	147	67
Gonorrhea	366	387	357
Granuloma inguinale	0	0	*
Lymphogranuloma venereum	10	0	*
Malaria	68	24	1
Measles	260	1647	1647
Meningitis, meningococcal	23	90	6
Meningitis, Pfeiffer-bacillus	3	5	3
Meningitis, pneumococcal	4	18	†
Meningitis, staphylococcal	0	0	†
Meningitis, streptococcal	0	0	†
Meningitis, other forms	4	2	†
Meningitis, undetermined	5	17	†
Mumps	2010	913	836
Pneumonia, lobar	300	646	618
Salmonella infections	3	1	5
Scarlet fever	1617	1200	1162
Syphilis	358	447	417
Tuberculosis, pulmonary	217	237	237
Tuberculosis, other forms	12	17	18
Typhoid fever	0	2	6
Undulant fever	2	6	2
Whooping cough	613	373	881

*Made reportable December, 1943.

†Pfeiffer-bacillus meningitis only other form reportable previous to 1941.

Comment

Bacillary dysentery, during January, showed a marked increase in the number of cases, reaching a figure more than three times that of December and seven times that of the seven-year median.

Scarlet fever reached the highest point for any January since 1933. The cases were well scattered throughout the Commonwealth.

Lobar pneumonia dropped to the lowest January figure since reporting began.

For the second time in history no cases of typhoid fever were reported in the month of January.

Geographical Distribution of Certain Diseases

Anterior poliomyelitis was reported from: Haverhill, 1; Lowell, 1; total, 2.

Anthrax was reported from: Lynn, 1; Peabody, 1; total 2.

Diphtheria was reported from: Barnstable, 1; Boston, 3; Foxboro, 1; Hull, 1; Mattapoisett, 1; Medford, 1; New Bedford, 1; Somerville, 9; Southbridge, 1; Waltham, 1; total, 20.

Dysentery, bacillary, was reported from: Beverly, 3; Boston, 7; Cambridge, 3; Lexington, 19; Malden, 2; Quincy, 1; Revere, 1; Wakefield, 4; Walpole, 1; West Boylston, 2; total, 43.

Encephalitis, infectious, was reported from: Chelsea, 1; Everett, 2; Somerville, 1; total, 4.

Malaria was reported from: Boston, 4; Camp Edwards, 8; Cushing General Hospital, 14; Haverhill, 4; Hingham, 1; Lowell, 1; Medford, 1; Melrose, 1; Quincy, 1; Regional Hospital, Waltham, 31; Somerville, 1; Webster, 1; total, 68.

Meningitis, meningococcal, was reported from: Boston, 8; Chelsea, 1; Chicopee, 1; Fall River, 1; Fitchburg, 1; Haverhill, 2; Lynn, 1; Marblehead, 1; Medford, 1; Naval Training School (Harvard University), 1; Plymouth, 1; Quincy, 1; Revere, 1; Wakefield, 1; Worcester, 1; total, 23.

Meningitis, Pfeiffer-bacillus, was reported from: Boston, 1; Bourne, 1; Salem, 1; total, 3.

Meningitis, pneumococcal, was reported from: Newton, 2; Springfield, 1; Worcester, 1; total, 4.

Meningitis, other forms, was reported from: Boston, 1; Lynn, 1; Tisbury, 1; Watertown, 1; total, 4.

Meningitis, undetermined, was reported from: Boston, 2; Chelsea, 2; Monson, 1; total, 5.

Salmonella infections were reported from Arlington, 1; Cambridge, 1; Melrose, 1; total, 3.

Septic sore throat was reported from: Attleboro, 1; Boston, 19; Everett, 1; Merrimac, 6; Millbury, 1; Norwood, 1; Watertown, 1; Whitman, 1; total, 31.

Tetanus was reported from: Grafton, 1; total, 1.

Trichinosis was reported from: Becket, 1; Boston, 4; Chelsea, 1; Shirley, 1; total, 7.

Undulant fever was reported from: Amesbury, 1; Boston, 1; total, 2.

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Clinic Consultant
Lowell	March 2	Albert H. Brewster
Salem	March 5	Paul W. Hugenberger
Haverhill	March 7	William T. Green
Brockton	March 15	George W. Van Gorder
Worcester	March 16	John W. O'Meara
Pittsfield	March 19	Frank A. Slowick
Springfield	March 21	Garry deN. Hough, Jr.
Fall River	March 26	Eugene A. McCarthy
Hyannis	March 27	Paul L. Norton

MISCELLANY

ABSENTEE SICKNESS RATE SOARS

Absenteeism due to sickness among male industrial workers showed a substantial increase for the third quarter of 1944, shooting the total industrial sickness rate for the year up to 37 per cent above average for the period 1935–1944, according to statistics released by the Industrial Hygiene Division of the United States Public Health Service.

Nonrespiratory-nondigestive diseases rose 15 per cent above the same period in the previous year. Conditions contributing to this record included a 26 per cent increase in "rheumatism," a 29 per cent increase in neurasthenia and other diseases of nervous or mental origin and a 34 per cent increase in diseases of the genitourinary system. The incidences of rheumatic diseases, diseases of the heart and arteries, nephritis and nervous diseases have never been equaled or exceeded within the past decade. Nervous

diseases showed the highest rate of increase, rising 76 per cent above the ten-year mean. The rate of respiratory diseases, although slightly lower than that in 1943, was 32 per cent above average for the decade. Digestive diseases also rose to a rate exceeding anything experienced within the ten-year period.

Employment conditions peculiar to wartime are held responsible. Categories of disease showing the highest increase involve mainly older workers. The hiring of workers long unemployed or retired is one factor held to contribute to the record sickness rate, as is the employment of youths and other inexperienced personnel, and the necessity for employing in industry men rejected by the armed services. Wartime working and living conditions are also thought to be reflected, since other factors held accountable include emotional strain and personal mental conflict, overcrowding in plants and war communities, fatigue due to the lengthened work week, and night work. — Reprinted from *Industrial Hygiene News Letter* (January, 1945).

CORRESPONDENCE

PHYSICIANS REGISTERED IN NOVEMBER

To the Editor: Attached is a list of the physicians registered by examination by the Massachusetts Board of Registration in Medicine on November 17, 1944.

H. QUIMBY GALLUPE, *Secretary*

* * *

Addes, Jacob (Middlesex University, 1944): 5123 14th Avenue, Brooklyn, New York.

Agrin, Irving Nelson (Chicago Medical School, 1943): 3053 36th Street, Astoria, Long Island, New York.

Becker, William (Chicago Medical School, 1943): 404 Hart Street, Brooklyn, New York.

Berkowitz, Moe (Middlesex University, 1943): 1319 Avenue P, Brooklyn, New York.

Boller, Raymond Jerome (New York University, 1942): 51-11 63rd Street, Woodside, Long Island, New York.

Camelio, Albert Patrick (Boston College of Physicians and Surgeons, 1944): Holy Family Hospital, Brooklyn, New York.

Coggan, Manuel (Middlesex University, 1943): 84 Mt. Vernon Street, Malden.

Dorsey, Joseph Farrell (University of Tennessee, 1937): 1221 Forrest Avenue, Memphis, Tennessee.

Ehrenberg, Bernard (Chicago Medical School, 1943): 632 Union Avenue, Elizabeth, New Jersey.

Eyres, Alfred Ernest (State University of Iowa, 1932): Worcester State Hospital, Worcester.

Fitch, Edna Mae Jones (University of Wisconsin, 1943): Sturgis, South Dakota.

Gelbin, Milton Ruel (Chicago Medical School, 1943): Barnet Memorial Hospital, Paterson, New Jersey.

Goldstein, Max Richard (Hahnemann Medical School, 1943): 605 Commonwealth Avenue, Boston.

Henry, Herman Gordon (Philadelphia College of Osteopathy, 1943): 35 W. Main Street, North East, Pennsylvania.

Hindle, Joseph Anthony (Jefferson Medical College, 1940): Boston Psychopathic Hospital, 74 Fenwood Road, Boston.

Hughes, Paul William (College of Physicians and Surgeons, Boston, 1944): 936 N. Irving Avenue, Scranton, Pennsylvania.

Jacobs, John Lesh (Harvard Medical School, 1929): West Townsend, Vermont.

Kennedy, Raymond James (Loyola University, 1926): 509 Third Avenue, Joliet, Illinois.

Lazarus, Sydney Simon (Chicago Medical School, 1943): 300 Ocean Parkway, Brooklyn, New York.

Levenson, Stanley Mortimer (Harvard Medical School, 1941): Boston City Hospital, Boston.

Loebel, Arthur Louis (Chicago Medical School, 1943): 1690 President Street, Brooklyn, New York.

Meyer, Manfried (University of Halle-Wittenberg, 1920): 41 Waltham Street, Boston.

Moldin, Sidney (Middlesex University, 1944): 141 E. Ravenwood Avenue, Youngstown, Ohio.

Murray, Oscar Beryl (University of Georgia, 1933): 605 Commonwealth Avenue, Boston.

Pross, Philip (Middlesex University, 1944): Wilmington General Hospital, Wilmington, Delaware.

Rettew, Philip Lewis (University of Pennsylvania, 1941): 605 Commonwealth Avenue, Boston.

Ross, Jasper Newton (University of Illinois, 1941): 17 Bowker Street, Brookline 46.

Schnall, Meyer DeWitt (Johns Hopkins University, 1942): 810 Madison Avenue, Bridgeport, Connecticut.

Schwartz, Oscar (Chicago Medical School, 1943): 1020 45th Street, Brooklyn, New York.

Sherwood, Harold R. (University of Arkansas, 1939): 605 Commonwealth Avenue, Boston.

Smoley, Melvin (Chicago Medical School, 1943): 212 West 22d Street, New York City.

Svenson, Orvas (Harvard Medical School, 1937): 2 Netherlands Road, Brookline.

Tauber, Hans (Middlesex University, 1943): 87 Hamilton Place, New York City.

Trevor, William (University of Maryland, 1940): 186 Commonwealth Avenue, Boston 16.

Turtletaub, George Nyles (Middlesex University, 1944): 1505 Ocean Avenue, Brooklyn, New York.

Waldman, Hyman (Middlesex University, 1944): 76 Verndale Street, Brookline.

Walker, Richard Parish (University of Tennessee, 1937): 37 Carlton Street, Brookline.

Wein, Eber Abraham (Chicago Medical School, 1943): 565 Manhattan Avenue, New York City.

Weissman, Morris Paul (Middlesex University, 1942): 1395 Eastern Parkway, Brooklyn, New York.

Woronoff, Morris (Middlesex University, 1943): 570 Lefferts Avenue, Brooklyn, New York.

NOTICES

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, MARCH 8

FRIDAY, MARCH 9

*9:00–10:00 a.m. Protein Nutrition in Problems of Medical Interest. Dr. Fredrick J. Stare. Joseph H. Pratt Diagnostic Hospital.

10:50 a.m. Tuberculosis of the Skin. Dr. G. Morris. (Postgraduate clinic in dermatology and syphilology.) Amphitheater, Mallory Building, Boston City Hospital.

12:00 m. to 1:00 p.m. Clinicopathological conference (Boston Floating Hospital). Joseph H. Pratt Diagnostic Hospital.

SATURDAY, MARCH 10

*10:00 a.m.–12:00 m. Medical staff rounds. Peter Bent Brigham Hospital.

MONDAY, MARCH 12

*12:00 m.–1:00 p.m. Clinicopathological conference. Peter Bent Brigham Hospital.

TUESDAY, MARCH 13

*12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital.

WEDNESDAY, MARCH 14

*9:00–10:00 a.m. The Glaucoma Problem and General Medicine. Dr. Joseph Igersheimer. Joseph H. Pratt Diagnostic Hospital.

*12:00 m. Clinicopathological conference. Children's Hospital.

*12:00 m.–1:00 p.m. Clinicopathological conference, Cambridge Hospital.

7:15 p.m. Graduate seminar in pediatrics. Children's Medical Service. Amphitheater 3A, Massachusetts General Hospital.

*Open to the medical profession.

MARCH 8. Certain Neurological Problems as Regards Children: Diagnosis and treatment. Dr. Franc D. Ingraham. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

MARCH 14. New England Pediatric Society.

APRIL 30. New York Institute of Clinical Oral Pathology. Page 120, issue of January 25.

MAY 22-24. Hotel Statler, Boston. Annual meeting of the Massachusetts Medical Society.

SEPTEMBER 17. American Public Health Association. Page 752, issue of November 30.

DISTRICT MEDICAL SOCIETIES

PLYMOUTH

MARCH 15. Goddard Hospital, Brockton.
APRIL 26. Toll House, Whitman.
MAY 17. Lakeville Sanatorium, Lakeville.

All meetings will be held at 11 a.m.

WORCESTER

MARCH 14. Worcester Memorial Hospital.
APRIL 11. Hahnemann Hospital, Worcester.
MAY 9. Annual meeting.